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CHAPTER CVII

DISEASES OF THE FACE AND JAWS

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Macrostoma.—A congenital widening of the mouth, due to non-union of the maxillary and mandibular processes. It may be unilateral but is usually bilateral and symmetrical. Other facial anomalies, such as auricular appendages are usually present. The normal limits of the mouth may be indicated by depressions showing the insertion of the orbicularis oris, muscle which constitute important landmarks in plastic operations designed to reduce the width of the mouth to normal.

Microstoma.—A congenital narrowing of the mouth due to a growing together of the tissues forming the lips. It is often associated with bony anomalies or defects of the soft parts. The treatment consists in making symmetrical transverse incisions sufficiently prolonged to restore the aperture of the mouth to a normal size. Mucous membrane flaps are then sutured to the raw surfaces to prevent subsequent union.

Facial clefts are due to lack of closure of the naso-orbital fissure. "They are usually unilateral, running from the usual situation of a hare-lip upward outside the nostril to the lower lid (Rose and Carliss)." They may involve the soft tissues alone or the bone, opening into the antrum of Highmore. Other congenital anomalies may be associated with facial clefts. The treatment consists in freshening the edges of the cleft and bringing them together with interrupted sutures. When the cleft involves the bone, the defect may be closed as in cleft-palate operations.

Macrocheilia.—Three types are known.

1. Congenital, is a thickening or hypertrophy of one or both lips; in this type there is hypertrophy of all the tissues of the lip, usually the lower lip, which is indurated, everted and thickened, exposing the lower teeth. The treatment is plastic removal. The condition is caused by localized lymphedema accompanied by proliferation of the connective tissue and may be accompanied by similar changes in the body of the tongue and surrounding tissue. The condition is painless, but the deformity is unsightly.

2. Acquired.—The acquired form occurs in tuberculous children and those living under poor hygienic surroundings and has been called strumous lip. Either lip may be involved, but usually the upper is affected. It may result from absorption from cracks and fissures in the course of chronic eczema or other dermatoses or during convalescence, from acute infectious diseases and in the cachexia consequent upon chronic infections, causing chronic inflammation and hypertrophy. It is also seen in mouth breathers, being caused by adenoids and hypertrophied tonsils.

Treatment.—The cause of the trouble must be investigated. Infected tonsils, adenoids, and carious teeth must be removed and dermatological lesions healed up. Improvement occurs but frequently the parts cannot be restored to normal because of connective tissue hyperplasia.

3. Syphilitic.—The syphilitic form occurs in the form of primary chancre from kissing; the use of infected china, glass, silver or pipe.

The classic lesion consists of a hard, slightly ulcerated elevated mass irregularly shaped, surrounded by an area of inflammatory tissue which may extend to the submaxillary glands. There may be a slight involvement of the surrounding cellular tissues. Involvement of the salivary and lymphatic glands is marked and the degree of absorption is greater than from genital lesions.

Primary chancre rarely requires differentiation because of its distinctive appearance. However, epithelioma may at times confuse but should be recognized by its slow growth, absence of discharge during its early appearance, the slow involvement of neighboring glands, the absence of acute inflammatory reaction, the lack of response to anti-syphilitic treatment and the Wassermann test. Epithelioma occurs more commonly upon the lower lip, chancre frequently upon the upper.

In the secondary stage the cutaneous lesions and mucous patches are characteristic. The late manifestations of syphilis present ulceration and gummata with diffuse induration of the lips, and extensive destruction of tissue.

Congenital syphilis is indicated by the presence of cracks, mucous patches, tubercles and cicatrices radiating from the mouth with the associated Hutchinson's triad.

Epulis.—Definition.—A fibrous tumor originating in the periosteum of the alveolus of the jaw.

Etiology.—It occurs most commonly in early adult life and frequently can be traced to some form of chronic irritation, most commonly a carious tooth. The lower jaw is more often affected.

Pathology.—The tumor appears around and between the teeth and resembles granulation tissue, though it does not bleed easily. It is usually irregular in outline and made up of a single mass but occasionally there are aggregations of smaller masses. The tumor is covered by mucous membrane and at times may undergo necrosis from interference with its blood supply by pressure upon the growing mass by the neighboring teeth. The tumor rarely reaches a large size unless malignant transformation occurs. It is composed largely of fibrous tissue. Cyst formation may occur.

Symptoms.—Usually due to pressure and mechanical interference with mastication.

Treatment.—Consists in the removal of any source of irritation followed by a thorough excision of the mass and curetting of its attachment. Should the tumor recur, malignancy is to be feared and a radical removal of the portion of the maxilla involved is indicated.

Malignant epulis will be considered under sarcoma of the jaw.

Necrosis of the Jaws.—**Etiology.**—Necrosis occurs more commonly in the lower maxilla than in any other bone and is due to the fact that practically all fractures of the lower jaw become compound and further to the septic condition of the mouth in individuals who neglect its care.

Necrosis may be caused by: (1) Trauma to the jaw, fractures, periostitis and osteomyelitis. (2) Infection from carious teeth involving the periosteum surrounding the alveolus and later the medullary cavity, such as pyorrhea alveolaris (Riff's disease). (3) Acute infectious diseases associated with noma and cancrum oris, as measles, scarlet fever, typhoid fever, acute septic endocarditis and pyemia. (4) Chronic infections, as tertiary syphilis, tuberculosis and actinomycosis. (5) Chemical poisons as mercury, lead and phosphorous. (6) Severe anemias and scurvy.

Pathology.—The pathology is that of an infective periostitis and ostmeyelitis. Miliary abscesses form in the medulla of the bone coalescing and forming larger collections. Necrosis of the periosteum corresponding to the area of osteomyelitis occurs. The pressure within the bone increasing, rapid destruction of tissue occurs until the pus finds an avenue of escape in the form of a sinus. One or several sinuses may later be found. The chronic irritation caused by the escape of the wound secretions leads to the development of masses of granulation tissue. In the periosteum, which remains alive, osteogenesis is stimulated and is manifested by the development of an involucrum surrounding a mass or sequestrum of necrotic bone of greater or less extent. The inflammation may extend into the soft tissues of the floor of the mouth or neck. The acute condition just described is met with only in virulent forms of infection.

Often infection occurs in the alveolus of a carious tooth leading to the formation of an alveolar abscess. The tooth may be elevated by the accumulation of pus and is exquisitely sensitive. The whole face upon the affected side becomes swollen, with the formation of a gum boil. Fluctuation may be detected in the gum about the tooth and if left alone the abscess may break. relieving the symptoms. In aggravated cases osteomyelitis occurs.

Chronic infection about the roots of one or more teeth, known as pyorrhea alveolaris (Riggs' disease), occurs most commonly in later years of life and is characterized by retraction and anemia of the gums, loosening of the affected teeth and usually a discharge of pus. Evidence is rapidly accumulating to prove the direct connection between local infections in the mouth and various disturbances formerly considered to be of a metabolic character, such as chronic rheumatism and arthritis deformans.

Symptoms.—The most severe manifestations occur in the acute septic form usually following trauma. Great swelling and tenderness with inability to open the mouth widely (inflammatory lockjaw) are followed by chills and fever. Saliva and pus may drip from the angles of the mouth and the breath is extremely foul. The tongue is heavily furred and attempts at examination are very painful. Leukocytosis is present and the urine may contain albumin and casts. Unless relieved by surgical measures the symptoms increase in severity until the pus finds an outlet by bursting through the abscess wall.

Gingivitis from lead poisoning occurs amongst painters and workers in lead. Its onset is usually slow and the first symptoms are colic and vomiting. Later the lead line appears upon the gums at the junction with the teeth, beneath which the mucosa is swollen and tender and bleeds easily. The teeth may become loose and fall out. Peripheral neuritis manifested by palsies, especially wrist-drop, frequently occurs. Secondary anemia is often pronounced.

Phosphorous necrosis formerly occurred among workers in match factories using yellow phosphorous where the fumes of phosphorous were inhaled. Fortunately, this is a rare condition at the present time because of the changes brought about by industrial medicine. In phosphorous necrosis inflammation usually begins about a carious tooth with enlargement of the bone. Suppuration with thickening of the bone and periosteum ensue with loosening of the teeth. It most commonly affects the lower jaw. In severe cases the whole lower jaw may be lost.

Tertiary syphilis in the form of a gummatous ostemyelitis and periostitis may cause necrosis of the jaws. It most commonly affects the upper maxilla and may be associated with perforation of the nasal septum or hard palate. It is usually not painful.

Tuberculosis of the jaws is infrequent and is usually secondary to tuberculosis of the soft tissues of the mouth. The process is similar to osseous tuberculosis elsewhere, *i.e.*, primary caries or rarefaction, formation of cold abscess which when evacuated either by incision aspiration or its own pressure becomes secondarily infected with pus organisms.

Actinomycosis of the jaw is rare. Although the infection is primary in a carious tooth it attacks the soft tissues most extensively and rarely invades the bone. The process is slow and mixed infection the rule. The diagnosis is usually made from the finding of the ray fungus in the lesions of the soft parts.

Scurvy (Barlow's Disease) is a constitutional disease due to nutritional disturbances characterized by subcutaneous hemorrhages especially over lower end of femur and upper part of tibia, hemorrhages from the nose, swollen, bleeding gums and progressive anemia.

The gums become bluish and little sacs containing blood form at the roots of the teeth which become loosened and may fall out. In some cases the swollen gums may cover the teeth. Hemorrhage from the gums takes place from the slightest trauma or even spontaneously. Salivation frequently occurs. The condition is frequently associated with rickets.

Treatment consists in the discontinuance of all sterilized and proprietary foods and the substitution of fresh cow's milk and fresh fruit, especially orange juice. When signs of inflammation appear much relief may be obtained by the application of an ice-bag to the neck and by holding chipped ice in the mouth. The patient should be fed through a tube and nutrition maintained.

As soon as evidence of pus is detected, free incisions should be made down to the bone. Necrosis of the bone may be noted by fragments of bone in the pus and by *x*-ray examination. When this is present the involucrum must be broken down, the sequestrum removed and the whole infected area thoroughly curetted and swabbed out with tincture of iodin. Usually this can be accomplished by an incision from within the mouth but where a large amount of bone is involved, an external incision may be necessary.

Cancrum Oris.—Definition.—An infective gangrene of the tissues making up the oral cavity, beginning on the gums or cheeks and causing extensive necrosis and sloughing.

Etiology.—Cancrum oris is seen rather frequently in the convalescence of acute exanthemata, especially measles, but also following pneumonia, erysipelas, nephritis, dysentery, scarlet and typhoid fevers. One-half the cases follow measles. It is rarely observed except in children and occurs in those weakened by unfavorable environment and poor food, especially in the tenement districts of large cities. Girls are more often affected than boys. It is encountered most commonly between the ages of two and five. Epidemics occur in orphan asylums and children's hospitals. Various pyogenic organism have been found in this infection, but the streptococcus is probably most often isolated. Usually a mixed infection develops with the streptococcus predominant.

Da Costa states that in noma positive Wassermann tests can be obtained. There is much discussion as to the specific cause of noma and as a consequence opinions differ as to whether or not it is to be regarded as contagious and requiring isolation. In view of the fact that epidemics occur in children's hospitals and institutions.

Pathology.—There is probably some slight atrium of infection in the buccal mucosa, although the lesion may escape attention. The mucosa of the cheek or gum becomes inflamed and later gangrenous. There is a central gray area of gangrene which sloughs, leaving a foul discharging surface. The surrounding tissues are invaded by the gangrenous process the whole cheek becomes enormously swollen and takes on a glossy-red appearance. Finally, the redness fades into a dull-black, indicating the formation of a slough, vesicles containing serum appear and perforation of the cheek may occur. An area of brawny induration surrounds and slough resulting from blocking of veins and lymphatics and cellular infiltration. In severe cases the gangrenous process may involve the whole cheek, tonsils and fauces, even destroying the maxillary bones and eyelids.

In mild cases a small slough forms upon the buccal mucosa. This either heals or ulcerates through the cheek leaving a fistula.

Symptoms.—Because of the virulence of the infection and the rapidity of absorption both locally and from the swallowed wound secretion the degree of sepsis is very great. Rigors or convulsions in young children may first call attention to the condition. The fever is high and the pulse rapid. Delirium may occur later, ending in coma. Pneumonia may supervene from aspiration of the septic wound discharges. The tissues are gangrenous, therefore there is but little pain and frequently the temperature is not high because absorption of the putrefactive material is delayed by the thrombosed vessels and infiltrated cellular tissue. The odor from the sloughing mass and from the patient's breath is sickening. The pulse is rapid and feeble.

Prognosis.—The mortality reaches 90 per cent.

Treatment.—This is often of no help because of the debilitated condition of the patient. Under an anesthetic all gangrenous tissue should be removed and the raw surfaces cauterized with the Paquelin cautery or nitric acid. When necrosis of the bones of the face has occurred, the sequestrum must be removed. Carious teeth should be extracted. "Rumple and Nicoll have used salversan, intravanous injections of 0.3 gr. at three-day intervals with benefit. Each injection produced a great local reaction in the cheeks" ("Da Costa's Surgery"). Careful attention to the hygiene of the mouth is necessary. Irrigation of the ulcerated surfaces with mild antiseptic solutions washes aways the foul secretions and promotes healing. The general nutrition of the child must be maintained at the highest possible point by correct feeding and tonics.

Where recovery ensues, plastic operations may be necessary to prevent deformities from scar contracture and to close defects.

Stomatitis.—Definition.—Inflammation of the mucous membrane of the mouth. It occurs under much the same conditions as gingivitis and frequently the two conditions are present at the same time.

Etiology.—The simple catarrhal form is usually caused by the ingestion of hot foods, corrosive chemicals or chronic irritation from carious teeth. It may follow dyspepsia and improper feeding in children. The mucosa is red and swollen, the tissues of the mouth take on a beefy appearance, and there is an increased production of saliva. Petechial hemorrhages may occur.

The ulcerative form is seen frequently in the course of acute infectious diseases, especially the exanthemata in children, in coma whether due to trauma or intoxication, pyemia, metallic poisoning—lead, phosphorous or mercury—and in debilitated states.

Pathology.—The ulceration frequently commences about the roots of carious teeth. The mucosa becomes swollen and painful and an area of discoloration surrounds the teeth. Frequently pyorrhea alveolaris is present. The breath is foul and salivation may be noted. The tongue is frequently greatly swollen and covered by a dirty yellow fur. Identations of the teeth upon the sides may be pronounced. The regional lymph glands may be enlarged.

Gangrenous stomatitis is discussed under noma. The local manifestations are usually over-shadowed by the causative state as in typoid. The treatment consists in the removal of sources of irritation as carious teeth and the treatment of the underlying condition. Chlorate of potash as a mouth wash or lemon juice and glycerin as a swab to the tongue are comforting to the patient and tend to loosen up and dissolve the dessicated epithelial crusts covering the tongue.

TUMORS OF THE JAWS

Osteoma.—(*A*) Compact osteomata are composed of masses of dense bony tissue and are spoken of as ivory exostoses. The mass is always sessile and usually situated at the angle of the jaw, but frequently attacks the wall of the external auditory meatus (Bland Sutton).

(*B*) Cancellous Osteomata resembles to a greater extent the normal structure of bone. Cartilage undergoing ossification may be found in them.

Symptoms are confined to pressure and mechanical disturbances, the tumor is not disseminated through neighboring structures and does not break down and ulcerate.

Treatment.—Consists in the removal of the growth when symptoms become severe.

There is no known etiology but chronic irritation from unerupted or carious teeth may play some part. Osteomata of the facial bones rarely reach a large size but because of their development upon a sessile base present considerable difficulty to operative removal. They are almost always singular and painless.

Carcinoma.—**Etiology.**—The tumor originates from the mucous membrane of the jaws, frequently at the site of a carious tooth. Often attention is first directed to the tumor in searching for a cause of the cervical adenitis which is a striking manifestation of the malignant invasion. In no other location upon the surface of the body are the regional lymph glands so conspicuously involved. When the tumor is made up of squamous epithelium it produces the typical epithelioma. The columnar celled variety is more rapid in the infiltration of surrounding tissues. As the mass grows, its contour is modified by the presence of teeth, between which it forces its way until it appears as granulation tissue. Its peculiar character has suggested the name of coxcomb (Da Costa). As ulceration and breaking down of the tumor proceed, the teeth become loose and fall out. The gums bleed easily, the breath is horribly foul, and the secretions of the mouth mixed with pus and fragments of cancer tissue drip away. Aspiration pneumonia is not uncommon in the stage of ulceration. The primary tumor never reaches the large size of sarcoma, hence symptoms due to mechanical obstruction are not common.

In carcinoma of the upper jaw the outer wall of the antrum of Highmore may be destroyed laying the interior open. This becomes more or less filled with the products of malignant degeneration and is very offensive.

Treatment.—Consists in the earliest treatment of any persistent nodule in the mouth. All sources of irritation must be removed, especially carious teeth. Dr. Charles Mayo has made the striking observation that the reaction of the oral cavity is acid in malignant disease. The normal reaction also is lost in pyorrhea alveolaris and other chronic infections, the inference being that carcinoma develops more readily in an acid medium.

Because of the early and widespread metastases, surgical treatment should be instituted as soon as the diagnosis is made. Radium is of great help in the early cases, and in those instances when radical operation is hopeless as it is frequently possible to heal over the local ulcerating process, thereby enabling the patient to take food without pain and to render the mouth free from the horrible fetor resulting from decomposing oral tissue. The patient may put on many pounds of weight and while the healing of the local lesion does not cure the patient it adds greatly to his comfort.

Odontoma.—Definition.—A tumor originating from the embryonal elements of teeth. The type is indicated according to the variety of tooth-germs involved. There are seven different varieties as described by Bland-Sutton ("Tumors Benign and Malignant," but only four are usually seen in man.

1. Epithelial Odontome (Fibrocystic Tumor).—This tumor arises from the enamel-organ and consists of a fibrous cyst wall containing a large series of smaller cysts separated by fibrous septa containing brownish fluid. Alveoli made up of aggregations of epithelial cells develop. The growth may reach large size and encroach on vital organs. It may be mistaken for sarcoma.

2. Follicular odontomes, more commonly known as dentigerous csyts. These fibrocystic swellings arise in connection with the development of permanent teeth and give rise to a remarkable appearance when bilateral. The cyst wall varies in size and is produced by the dilated tooth follicle. Crepitation may be present when the wall is thin. An unerupted tooth is usually found within the cyst cavity which also contains fluid. They are more common in the upper than the lower maxilla and frequently develop in the antrum of Highmore. Absence of one or more permanent teeth is seen. Fluctuation, egg-shell crackling and suppuration may occur.

3. **Radicular Odontome.**—This tumor takes its origin from the root of the tooth and is composed of cement and dentine. In its development the dental nerves may be compressed causing great pain.

4. **Composite odontoma** are made up of a mixture of enamel dentine and cement and develop in the maxillæ. They attain much larger size in the upper maxilla and are frequently confused with osteomata.

Symptoms.—These are mainly due to pressure, pain and to mechanical obstruction. When the second or third divisions of the fifth nerve are compressed by the tumor the pain is intense, but large odontomata may develop painlessly. Encroachment upon the naso-pharynx is common and the child may be looked upon as suffering from adenoids. The rapid increase in the growth of the tumor, however, in most cases rules out simple naso-pharyngeal obstruction. In the follicular type the peculiar egg-shell crackling may suggest sarcoma of the periosteum or bone and resections of the maxilla have been done under the diagnosis of malignancy. Careful roentgenologic study of the tumor or removal of a specimen for microscopic examination will clear up the diagnosis. The roentgenogram will frequently show aggregations of unerupted or misplaced teeth. Osteomyelitis and osteoma must be differentiated.

Treatment.—This is surgical and consists in the enucleation of the tumor when this can be done readily, or the breaking down of a portion of the cyst wall, curetting the contents out, swabbing with tincture of iodin and packing with gauze.

Dental Cysts.—These usually occur about the dead roots of permanent teeth and are said to be due to a stimulation of the primitive enamel organ by the constant irritation maintained by necrotic teeth. The upper bicuspids and molars are most commonly affected. The cyst varies in size from a pea to a pigeon's egg and is unilocular. It is attached to the apex of the root and develops at the expense of the bone causing an expansion of the maxilla. Infection of the cyst may occur by contamination with the septic contents of the carious tooth, giving rise to the added phenomena of inflammation. Usually, there is a painless swelling about a dead tooth which gradually enlarges and finally fluctuates. Below the cystic area the rather sharp margin of the bone can be felt. Egg-shell crackling is sometimes present. The tumor may invade the antrum of Highmore.

Symptoms.—Are usually only due to the encroachment of the cysts upon surrounding structures unless infection takes place.

Treatment.—Consists in removal of the carious tooth or root, curetting out of the cavity and packing with gauze.

Leontiasis Ossea.—This is a remarkable condition of unknown origin characterized by the outgrowth of osseous masses from the bones of the face.

It occurs in young adults and is thought by some to be caused by syphilis and rickets, but the connection remains in doubt.

The progress of the disease is slow, but at times the orbit or cranial cavity may be encroached upon producing pressure which may endanger life. Usually, however, the bony masses grow asymmetrically, destroying normal facial contour and expression without causing symptoms. Occasionally, the patient complains of tic douloureux.

Treatment.—Consists in relieving the symptoms caused by pressure by removing a portion of the tumor.

Sarcoma.—Sarcoma of the jaws may originate from the alveolar periosteum or from teeth follicles. The former is usually of the small round- or spindle-celled type and grows rapidly, causing in most cases great deformity and early involvement of the neighboring lymphatic glands. Sarcomata frequently originate within the antrum, causing great expansion before involvement of surrounding structures takes place. Growth of the tumor may involve the orbit, producing a unilateral exophthalmos. The lachrymal duct, nasal fossæ and oral cavity may be encroached upon causing death by mechanical means. Sarcoma of the jaws has been observed in infancy.

Symptoms.—Due to pressure upon the maxillary division of fifth cranial nerve and to obstruction of the respiratory and oral passages. Ulceration of soft parts may lead to death from aspiration pneumonia.

Differentiation is necessary from benign tumors of the jaws, especially odontomata and inflammatory masses.

Treatment.—Consists in resection of one-half of the jaws and the employment of the *x*-ray and radium. The use of Coley's fluid has apparently been of benefit in some cases.

FRACTURES

Fracture of the Superior Maxilla.—Etiology.—Direct violence is practically always the exciting factor, associated with injuries to the malar, lacrimal or maxillary bones, rarely in connection with fractures of the base of the skull.

Pathology.—Any of the processes of the superior maxilla may be broken off, namely, the nasal, alveolar, or orbital processes. The malar bone may be depressed into the maxilla and there impacted. These fractures are frequently compound, either upon the surface of the face or within the oral cavity. Hemorrhage may be very considerable because of the vascularity of the tissues involved. Surgical emphysema of the face and neck is frequently observed, when the fracture communicates with the upper respiratory passages, and may produce enormous swelling of the facial tissues with obliteration of the normal lines of expression. The patient becomes plum colored and the tissues of the head, neck and thorax merge. Necrosis of the bone is rare, because of the abundant blood supply.

Symptoms and Physical Signs.—There is marked deformity of the upper jaw especially when the malar or nasal bones are involved. A false point of motion may be elicited when the entire upper maxilla is detached but when impaction occurs in connection with involvement of the malar bones, crepitus and false point of motion are not always easily demonstrated. When the alveolar processes alone are fractured, a false point of motion may be demonstrated together with crepitus. There is a marked swelling of the tissues of the upper portion of the face due either to hemorrhage into the cellular tissues or to emphysema. Chewing is impossible because of the deformity or because of the mobility of the alveolar processes and pain. In compound fractures infection is the rule because of communication with the septic oral cavity. However, the vitality of these tissues is great because of their vascularity and ordinary infections are usually well tolerated. Erysipelas may develop when no definite lesion of the skin or mucous membrane can be demonstrated. Anesthesia of the area supplied by the infra-orbital nerve may follow fractures of the superior maxilla. Roentgenograms are frequently necessary in detecting this type of fracture when no deformity or crepitus can be demonstrated.

Treatment.—In simple fractures the depressed bone may be elevated by an incision through the mucosa on the inner side of the upper lip through which some type of elevator may be inserted. Pressure through the antrum may be necessary by means of an urethral sound introduced through an opening made through the canine fossa. Immobilization is best obtained by a four-tailed bandage about the jaws or by means of a plaster cast reaching well down onto the shoulder. Feeding is carried out through a rubber tube introduced behind the last molar tooth, when none of the teeth are missing.

Fracture of the Inferior Maxilla.—**Etiology.**—This is a comparatively common type of injury in adults but occurs only rarely in children and may be caused either by direct or indirect violence. More commonly the violence is applied to the point of the jaw tangentially, the maximum stress falling upon the condyle just behind the angle of the jaw. The greatest strain is usually felt upon the side opposite to which the violence is applied. Fracture by direct violence occurs as a result of gunshot wounds or blows.

Pathology.—Fractures of the lower maxilla while they may be simple at first are frequently converted into the compound type because of the thin layer of tissues overlying the bone. Frequently the mucosa or tissues of the face are lacerated by the trauma producing the fracture. Infection is the rule, it being impossible to render the oral cavity aseptic. However, as in fractures of the superior maxilla, infection is rarely dangerous because of the natural resistance of the tissues due to their great vascularity. The line of fracture usually is oblique and situated at or near the junction of the body with the ramus of the jaw just behind the angle. Usually only one side is fractured when the injury is the result of indirect trauma. In gunshot fractures portions of both rami or bodies may be carried away together with the teeth and portions of the soft tissues. (Fracture may also take place along the mental foramen or through the neck of the condyle.) Hemorrhages from fractures caused by direct violence may be very severe because of the extreme vascularity of the soft parts.

The deformity resulting is usually very apparent because of the prominence of the lower maxilla under normal conditions. There is rarely a fracture of the inferior maxilla without deformity.

Symptoms.—There may be no signs of local trauma in the fractures caused by indirect violence. In those caused by gunshot or direct injury part of the bone beneath the site of the injury may be laid bare together with the teeth. Crepitus and false point of motion are more readily detected in this situation than anywhere else, except in fractures of the clavicle. Inability to chew or to articulate freely, is present, while saliva drips from the angle of the mouth. When infection takes place the breath may become horribly fetid, and pus is mixed with the dripping saliva. When osteomyelitis occurs small spicules of bone may be discharged through the mouth or sinuses may appear upon the surface of the jaw. *X*-ray examination is always necessary in diagnosis of these fractures.

Treatment.—Reduction may frequently be accomplished without aid of a general anesthetic. However, where there is much deformity and where it is necessary to remove pieces of bone and soft tissue in open wounds, general or local anesthesia should be employed. In the reduction of the deformity the patient himself knows when the fragments are in accurate alignment because of the comfortable contact of the teeth resulting when the normal conditions have been re-established.

Immobilization may be secured by a four-tailed bandage, by wiring the teeth of the upper and lower maxillæ together, or by means of a plaster cast. The services of a dentist should be secured in order that a vulcanite plate may be obtained to fit into the floor of the mouth. This is secured either by means of wires passed between the last tooth and through perforations in the plate, or by means of dental cement. This provides a very firm and comfortable method of immobilization and enables the patient to at once begin mastication. Furthermore, it allows the patient to brush the teeth and to irrigate the oral cavity with an antiseptic solution. Union takes place in from four to five weeks. In cases of osteomyelitis it may be necessary at a later time to open up the involucrum and to remove a sequestrum of bone.

Fracture of the Nasal Bones.—**Etiology.**—The nasal bones are usually fractured as the result of direct violence and frequently in conjunction with fracture of the malar or upper maxilla. The line of fracture is usually transverse and may involve the frontal bone or the base of the skull. The deformity is marked, consisting of deviation to one side alone or combined with depression of the bridge of the nose.

Laceration of the nasal mucosa is the rule, resulting in hemorrhage and frequently infection from the septic contents of the nares. The cartilage may be detached or deviated especially in those fractures causing a saddle-shaped deformity. Because of the great sensitiveness of the nasal mucosa, reduction of the deformity must usually be done under a local or general anesthetic. An *x*-ray is of great help and should always be obtained. When the deformity merely consists of a deviation of the lower fragment without depression, reduction may be accomplished by means of a well-padded, long-bladed forceps; one blade of which is inserted into each nasal cavity, pressure being made upon the septum, while the fragments are manipulated into position. Where depression exists, there is frequently overlapping of the fragments and it is necessary to exert pressure from within combined with counter pressure from without to overcome the overriding and to reduce the deformity.

Immobilization is secured by the introduction of a hard rubber nasal splint in each side which fully distends the nasal passages, thereby supporting and immobilizing the fragments.

Severe hemorrhage from the nose, surgical emphysems and even cerebral symptoms may result from a fracture of the nasal bones. There is often a displacement of the septum, producing an obstruction.

Treatment.—The treatment is reduction of the fracture by means of forceps or elevator introduced through the nares and fixation of the fragments by means of a nasal splint.

Fracture of the Lacrimal Bones.—The lacrimal bones extend from the nasal bones to the lateral mass of the ethmoid and are rarely fractured alone but usually in combination with the nasal and malar bones. Fracture of the lacrimal bones produces an interference with the tear duct. Surgical emphysema often results. When the nasal bones are fractured, treatment consists of restoring the lumen of the duct when possible by the elevation of depressed fragments by the introduction of a probe. In severe cases the duct must be removed.

Etiology.—Fracture of the malar bone is usually associated with fractures of the other bones of the face. It produces a marked depression.

Fracture of the Zygoma.—Etiology.—The zygoma is usually fractured as the result of direct violence. There is a marked depression produced, vertical displacement is limited because of the attachment of the temporal and masseter muscles. Interference with the movements of the lower jaw may occur when the depressed fragment impinges upon the condyle. Treatment consists in the elevation of the depressed fragment either by pressure from within the mouth or by elevating the fragment by means of a hook passed through a small incision above the fracture. The deformity does not tend to recur when reduction is complete.

Fractures of the Malar Bone.—Fracture of the malar bone are usually associated with fracture of the zygoma or nasal bones. Occasionally it occurs alone when produced by direct violence. It is rare in children and even in adults it is frequently unrecognized.

When the bone is depressed a hollow may be seen and palpated, just below and to the outer side of the orbit. When the depressed bone or fragment carries with it a portion of the zygoma, movements of the lower jaw are interfered with. Hemorrhage into the tissues in and about the orbit with pressure upon the infra-orbital nerve may occur.

Treatment consists in the elevation of the depressed bone either by pressure from within the mouth or by an incision above the bone and elevation by means of a lever or by hooks. There is little tendency to recurrence of the deformity when it has been once reduced and no fixation appliances are required.

AFFECTIONS OF THE TEMPOROMAXILLARY JOINT

Anatomy.—The intra-articular cartilage rests in a shallow recess. There is a separate synovial membrane on each surface of the cartilage. External and internal lateral ligaments hold the condyle firmly in the glenoid fossa, assisted by the action of the temporal, masseter and external pterygoid muscles. The nerve supply is from the third division of the fifth cranial nerve, which also gives branches to the teeth of the lower jaw. For this reason pain is referred to the temporomaxillary from tooth affections with freqently spasmodic closure of the jaw.

Acute Synovitis.—Etiology.—Acute synovitis may occur in the course of acute articular rheumatism, tonsillitis, as an extension from inflammation, hyper-extension of lower jaw, trauma to the joint directly, or indirectly, or apparently without any definite etiologic factor. The synovial membrane becomes acutely inflamed, resulting in hypersecretion of synovial fluid, constituting a hydrops of the joint. This may be manifested clinically by limitation of movement and fluctuation. There is pain on movements of the jaws and a swelling may be noticed about the joint. The patient cannot chew solid food. On palpation swelling and fluctuation may be noted but the contents of the joint rarely become septic unless contaminated from without, as by aspiration with an infected needle. When infection takes place the local manifestations become much more acute, the skin is red and hot, fluctuation and tenderness may be elicited by palpation, all movements are painful and constitutional symptoms indicative of absorption of toxic products supervene.

Treatment.—This consists in rest and immobilization by means of a four-tailed bandage, and aspiration of the contents of the joint if the swelling becomes large. Counterirritation by blisters, application of tincture of iodin are useful. The usual constitutional treatment for acute articular rheumatism or tonsillitis should be instituted. The injection of 2 per cent. formalin solution in glycerin is very painful and its use is not warranted.

Acute Septic Arthritis of the Temporomaxillary Joint.—Etiology.—Acute septic arthritis of the temporo-maxillary joint may occur as a sequel to acute simple synovitis or in the course or as a sequel to acute exanthemata, especially measles. It has been observed following tonsillitis, pyorrhea alveolaris, pneumonia, typhoid, scarlet fever, acute specific urethritis, and as a result of general sepsis or tuberculosis.

Extension of the septic process in osteomyelitis of the jaw may involve the temporo-maxillary articulation with rapid destruction of the joint. Penetrating injuries, as knife and gunshot wounds of the periarticular tissues and compound fracture offer an opportunity for the entrance of pathogenic organisms.

Pathology.—The infection rapidly destroys the lining endothelial cells leaving a raw abraded surface of connective tissue. There are the usual phenomena of inflammation as a result of the rapid growth of the pathogenic flora and a hypersecretion of serum and synovial fluid. A fibrinous exudate cements adjacent areas of synovial membrane and serves to limit the spread of infection and to minimize the amount of absorption. The interarticular fibro-cartilage is destroyed and the tension of the accumulation of septic products within the joint becomes greater until the pus is liberated by incision or bursts through the periarticular tissue. The fibrinous exudate becomes organised into firm connective tissue bands limiting movement of the jaws while true bony union may occur between articular surfaces. There is frequently thickening of the periarticular tissues and deformity may occur from scar contraction. While suppuration within the temporo-maxillary articulation may occur alone, it is more commonly part of a polyarticular involvement. Bilateral arthritis may occur.

Symptoms.—It may be manifested by chill or convulsions in children, high temperature, pain, swelling and redness about the joint. There may be a grating sensation upon moving the jaws. The diagnosis usually rests upon the recognition of septic infections in other joints.

Treatment.—Consists in the removal of the original source of infection if possible, the aspiration of the joint, and early immobilization to prevent fibrous or bony ankylosis.

Ankylosis of the Temporomaxillary Joint.—**Etiology.**—Results from acute infections, tuberculosis, arthritis deformans, or penetrating wounds. Involvement of the soft tissues surrounding the joint may result in thickening and contractures of the soft parts, constituting a fibrous ankylosis. Extensive burns of the face may result in contracting scars which will prevent movements of the jaws.

Symptoms.—There is a gradually diminishing ability of the patient to open the jaws, often accompanied by pain. Tenderness on pressure may be noted over the joint. Bony ankylosis rarely occurs in both joints.

Diagnosis.—True ankylosis must be differentiated from hysteria and spasms caused by the irritation of carious teeth or painful conditions about the oral cavity.

Treatment.—Prophylactic care of burns and wounds affecting the soft parts or joints is most necessary in preventing subsequent fibrous ankylosis. Once, however, it is established separation of the contracting areas and plastic operations upon the skin are frequently necessary. Flaps of skin for the face are usually best taken from the forearm or arm by the pedicle method. The resection of the condyle or osteotomy with the formation of a pseudoarthrosis upon the anterior border of the ramus or the angle of the jaw may be necessary even after the contracting fibrous tissue has been cut. Arthroplasty for bony ankylosis has been successfully done and consists in the separation of the bony surfaces by a chisel and the insertion between the articular surfaces of a thick flap of the temporal muscle and fascia so arranged as to preserve its blood supply and to prevent traction upon the flap. Early mobilization of the joint must be brought about as soon as possible. If the nutrition of the flap can be preserved a useful joint is likely to result. Resection of the condyle and a portion of the neck is a satisfactory method especially in ankylosis of both joints.

Lockjaw.—**Etiology.**—This curious condition is frequently observed after trauma to the jaws incident to the extraction of teeth, in acute inflammation of the temporomaxillary joint and soft tissues, abscesses, periostitis, osteomyelitis and fracture or dislocation of the jaws. Injuries and inflammation in the parotid region, as abscesses and mumps, or tumors of the parotid gland, may cause inability to open the jaws. Destructive processes of the tissues of the face, as concrum oris, sarcoma of the jaws, burns, bullet or lacerating wounds, syphilis, tuberculosis and actinomycosis resulting in scar contraction may prevent movement by mechanical interference. Diseases of the nervous system, as tetanus and hysteria, may produce inability to open the jaws because of the tonic contraction of the muscles.

Treatment.—Depends upon the cause. Those cases caused by inflammation or irritation of the branches of the fifth cranial nerve recover when the source of the trouble is removed. Tonic spasm of the muscles of the jaws is treated by a sedative, general or spinal anesthesia in connection with antitetanic serum and local exposure at the site of the atrium of infection. Resection of the condyles or arthroplasty will usually give a movable joint in bony ankylosis. Resection of cicatricial bands combined with plastic operations upon the soft tissues will frequently restore the mobility when the scar interferes with function. In hysterical joints the controlling idea of the patient should receive appropriate treatment.

Dislocation of the Temporomaxillary Joint.—The articular eminence of the temporomaxillary joint limits extension of the lower jaw. However, in forcible movements the condyles may slide past the eminentia and be forced into the zygomatic fossæ. Although the articular eminence is not developed until puberty, dislocations of the jaw are more commonly observed in the adult than in children. The usual dislocation is bilateral and forward. Dislocation in a backward direction is always accompanied by a fracture of the anterior wall of the auditory canal. Dislocation upward is very rare, only one case having been reported, in which the condyle was forced through the articular surface into the cranial cavity. Four cases have been reported of dislocation in an outward direction.

Etiology.—Ingestion of large masses of food, or falls or blows upon the chin with the mouth open may produce a dislocation. Forcible manipulation about the mouth, as in the extraction of impacted teeth while the patient is under general anesthesia has caused dislocation. Because of the consequent relaxation of the ligaments one dislocation may predispose to subsequent luxations until in some persons the least attempt to open the mouth widely will produce dislocation. Middle-aged women most commonly are affected by this accident.

Symptoms.—The patient presents a striking appearance. The mouth is held open, the chin and lower jaw project forward and are held in a fixed position, the mouth cannot be closed, and saliva dribbles constantly from the angles of the mouth. There is a depression in front of the ear corresponding to the empty glenoid cavity. The condyle frequently can be felt below the zygoma either from without or by the palpating finger within the oral cavity. The massester and temporal muscles are tense. It is impossible for the patient to chew and articulation is difficult, especially the pronunciation of the labials. Pain is present from pressure on the branches of the fifth cranial nerve. In a unilateral dislocation the chin deviates to the sound side.

Treatment.—Most cases may be reduced without the aid of a general anesthetic. It is necessary to depress the condyle below the level of the eminentia articularis to restore the head to the glenoid fossa. This is done by protecting the thumbs of the operator by thick bandages, having the patient sit in a straight-backed chair while strong downward pressure is made upon the lower molars by means of the thumbs introduced into the mouth. By this manoeuver the condyles are made to slide past the eminentiar articularis and the contraction of the massester and temporal muscles pulls the condyles into the glenoid cavity. After reduction the jaw should be immobilized by a four-tailed bandage for a week or 10 days, the patient meantime being fed by means of a rubber tube passed behind the teeth. In old cases reduction as in other joints is extremely difficult. A general anesthesia is required and forcible manipulations are necessary for the purpose of breaking up the old adhesions. Failing in this method of reduction open operation is necessary.

An incision is made over the joint and an effort made to free the condyle from its position anterior to the eminentiæ articularis. In many cases it is impossible to mobilize the bones sufficiently to bring about the reposition of the condyles in the glenoid cavity. Under these circumstances resection of the condyle may be necessary. The McGraw method consists of the introduction of a steel hook through a longitudinal incision over the temporo-maxillary articulation, an attempt being made to catch the condyle in the hook and replace it in the glenoid fossa.

ACUTE OSTEOMYELITIS OF THE JAWS

Osteomyelitis may be regarded as a pyemia, manifested clinically by local suppuration of the bone and periosteum and constitutionally by symptoms due to the presence of the infecting organisms or their products in the blood.

Etiology.—From an anatomic standpoint there are certain conditions which predispose to the arrest and localization of the infective material in the bone. The blood vessels in the region of the epiphysis are of an embryonal character. They are irregular in their course and lumen, favoring stasis and arrest of any foreign material in the circulation. The bone adjacent to the epiphysis is composed of spongy tissue.

The importance of trauma to a tissue so naturally fitted to undergo pathologic change cannot be over-estimated. The nutrition of the part is poor, especially in young persons up to 20 years of age. The trauma which initiates osteomyelitis may have been so slight as to have been forgotten by the patient, but upon careful questioning, the history of an injury to the part may frequently be obtained. The degree of trauma may vary from a simple contusion of the skin to a compound fracture or penetrating injury, or the osteomyelitis may follow a prolonged irritation of the bone, as by a carious tooth. In the case of open wounds the infective material is usually introduced from without in contra-distinction to the subcutaneous wounds in which the infection is of hematogenous origin. Exposure to cold has long been regarded as an important causative factor in osteomyelitis and there can be no doubt that chilling of the surface aids stasis and tends to venous thrombosis.

Osteomyelitis is commonly observed as a complication or sequence of acute infectious diseases. It may occur during or after typhoid, pneumonia, scarlatina, measles, variola, diphtheria, pyemia and puerperal septicemia. The fetus has been infected from the blood of the mother suffering from rheumatism and acute ulcerative endocarditis (Elingart). The newborn child has suffered from acute osteomyelitis, the result of an infection of the umbilical cord (D'Astros).

As the disease occurs during the period of most active bone proliferation and in the embryonal tissue of the metaphysis, the greatest incidence of osteomyelitis is found between the ages of 8 and 17. Osteomyelitis of hematogenous origin only occurs in 3 per cent. of cases after ossification is complete (Trendel).

Epidemics have occurred in certain localities, very likely following infections by the tonsils or upper respiratory tract.

The infecting organisms may enter the blood stream through a small abrasion of the skin, superficial burns, dermatological lesions, especially eczema, furunculosis, phlegmon, and erysipelas. Angina, catarrhal bronchitis and enteritis in children may provide an entrance (von Bruns).

It is interesting to note in the consideration of the bacteriology of osteomyelitis that each type of pathologic microörganism preserves its individuality and plays its rôle as characteristically in the osseous as in the soft tissues. In infections by the bacillus typhosus, no acute purulent condition is produced unless a mixed infection occur at the point of inflammation. A typhoid abscess, which, when not contaminated, takes on many of the clinical characteristics of a cold abscess, becomes the typical process of acute infective osteomyelitis when there is a mixed infection with pyogenic organisms. The typhoid form of osteomyelitis tends to undergo resorption and encapsulation or to become chronic when not contaminated with other organisms. Infections with the staphylococcus aureus occur in cases of moderate severity, while the streptococcus produces a rapid necrosis of the bone and is frequently fatal. In an analysis of 78 cases of acute osteomyelitis by Trendel, in von Bruns clinic the staphylococcus pyogenes aureus was present in 66; the streptococcus in 5; staphylococcus and streptococcus five times; and the diplococcus lanceolatus of Fraenkel, once.

Pathology.—The simplest form of acute osteomyelitis occurs in every fracture, as evidenced by cellular proliferation, extravasation of blood, and callus formation at the point of injury. In acute infectious osteomyelitis the introduction and retention of the pathogenic microörganisms occur either at the site of the trauma to the bone or periosteum in open wounds, as a metastatic focus from a suppurative process elsewhere, as sequelæ of some acute infectious disease, or finally, as a hematogenous infection, the point of localization being determined by a previous trauma or by the arrest and proliferation of the pathologic flora in the terminal capillary loops of the metaphysis of the growing bone.

Pathogenesis.—The infection may begin in the periosteum or medulla, rarely in the spongy tissue or compact bone.

(*a*) In the periosteum the process begins as a hyperemia and thickening, followed by a purulent infiltration beneath and around the periosteum, burrowing into the soft tissues and elevating the edematous and necrotic membrane from the bone. In severe cases there is a complete necrosis of the shaft of the bone, corresponding to the area of periosteal involvement, while in less purulent infections the process may subside with the production of a simple osteoporosis.

The tension of the infective material beneath the thickened periosteum may cause it to enter the Haversian canals and infect the medullary cavity, producing a rapid necrosis of the entire bone.

In other cases, the purulent material reaches the surface and produces a discharging sinus, or it may travel along the muscular and fascial planes and reach the periarticular tissues, finally causing a septic arthritis of the contiguous joint. When a limited portion of the periosteum is destroyed, necrosis of the underlying bone corresponding to the periosteal involvement occurs. In the process of bone regeneration which follows, the necrotic bone is not usually absorbed, but is separated from the healthier tissue and remains as a foreign body or sequestrum, frequently retaining pathogenic microörganisms in its substance for many years which may remain quiescent for long periods of time, only to break out again in the same or different bones after a slight trauma or other predisposing influence. The germinal layer of the periosteum produces an enveloping sheath of bone, the involucrum, which surrounds the sequestrum more or less completely, depending upon the condition of the periosteum. This involucrum is frequently honeycombed by sinuses called cloaca leading from the invested sequestrum to the surface.

(*b*) In the medullary cavity the introduction of the infection is followed by hyperemia, cellular proliferation, and the exudation of a serous, hemorrhagic or purulent material, which forms grayish-yellow foci in the marrow, resulting in various sized abscesses which may remain discrete or coalesce, converting the medullary space into a pus cavity. The tension under which the retained purulent material is held forces the infection into the Haversian canals and bone spaces, from which it communciates with the periosteum.

The involvement of the epiphysis or end of the diaphysis may result in necrosis of the intermediary cartilage and epiphyseolysis with or without involvement of the neighboring joint. In cases of low virulence or in cases in which the tension within the medulla has been relieved by an opening in the shaft of the bone, the destructive process confines itself to the medullary cavity, producing a necrosis of the internal layers of the bone without involvement of the periosteum or epiphyseal cartilage.

In the rare cases of osteomyelitis albuminosa, decribed first by Poncet and Ollier and later by von Bergmann, there is a profuse subperiosteal collection of synovial-like fluid, causing the clinical manifestations of pain, swelling, and fever. The process occasionally attacks the medulla in the region of the metaphysis. There is no pus formation and von Bergmann considers it to be the result of the action of attenuated pus cocci or a late manifestation of syphilis or tuberculosis. The condition tends to become chronic, with sinus formation and bone necrosis.

The process of repair in acute infective osteomyelitis is essentially the same as the primary growth of bone or as the healing of fractures modified by the presence of an acute infective process.

The period of incubation lasts from a few hours to a few days. This of course, can be reckoned only in those cases where there is a history of trauma, and lasts from the time of injury to the onset of symptoms.

Symptoms.—These vary with the variety and virulence of the infective organisms and the type of trauma. In severe forms the local manifestations are rapidly overshadowed by the constitutional symptoms. Pain is the dominant symptom. It appears first and as a rule lasts throughout the disease, increasing in intensity until the tension of the infective material confined in the medullary cavity or beneath the periosteum, to which the pain is due, is relieved by incision and removal of a portion of the osseous wall or by complete necrosis of the part. The pain radiates to the joint and may simulate tic douloureux.

Chill: This sometimes precedes the pain in time but not in importance. It comes on abruptly and is due to the rapid absorption of the infecting microörganisms and their products into the circulating blood. The high tension under which this rapidly increasing pathogenic flora is held within the unyielding confines of the bone favors septic embolism and multiple foci of suppuration, the invasion of fresh areas being manifested by renewed rigors.

Fever, of a high, angular, septic type is always present. It is caused by the absorption from the area of infection and continues until the source is removed. Accompanying the fever may be signs of acute general intoxication, namely, flushing of the skin, dry, coated tongue, scanty, albuminous urine, headache, and frequently, nausea and vomiting. The blood shows also a high polymorphonuclear leukocytosis. Cultures made from the blood within 24 hours of the initial chill frequently show the infective microörganisms.

Diarrhea with 5 to 10 dark movements daily is common in children.

Splenic enlargement may be due to the general pyemia or to infarction by the invading organisms. The spleen is palpable clinically in a large number of cases and is frequently tender to the touch.

Scarlatiniform, morbilliform and urticarial eruptions may occur, resembling antitoxin rashes. Herpetic, vesicular, and pustular lesions have been noted.

In severe cases convulsions and delirium, coma, and status typhosus may supervene. Occasionally, meningitic symptoms are observed.

The local findings are as follows:

Swelling is noticed during the first 24 hours after the initial pain and chill. It is fusiform in shape and may extend the length of the bone. When the periosteum is destroyed and the pus burrows along the soft tissues to the periarticular structures and into the joint cavity, the picture may be one of acute spreading infection.

Redness of the overlying skin due to the local inflammation is noticed. Tenderness over the affected area is exquisite and is due to the tension under which the infective material is held, the patient being uable to bear the slightest pressure upon the part. Movements of the temporo-maxillary joint may be impossible and when present cause great pain. Edema and fluctuation of the soft tissues occur later and pus may burst through the skin and produce a discharging sinus. Loss of function is always present and may be due to the intense pain, arthritis of the temporo-maxillary joint, necrosis of the body or ramus of the jaw, epiphyseolysis, spontaneous fracture of a subluxation of the head of the bone. The *x*-ray is of value in confirming the above changes and also for showing the osteoporosis, sequestrum formation and abscess cavities.

Clinical Course.—(1) **Severe Form.**—In this type, the pyemia and constitutional symptoms predominate. The onset is sudden, with acute pain in the body or ramus of the jaw, rigor, high fever, rapid, weak, irregular pulse, and convulsions in young children.

2. **Mild Form.**—Both the local and systemic symptoms are slight and usually subside upon evacuation of the abscess.

3. **Usual or Moderate Form.**—The local manifestations predominate, although the onset may be accompanied by a severe general reaction. This type of osteomyelitis of the jaw tends to become chronic and is frequently followed by sequestrum formation, ankylosis and bony deformity. However, the extent of medullary or periosteal involvement is no index to the severity of symptoms of the virulence of the infecting organisms.

Terminations and Complications.—(1) **Complete recovery** is possible without destruction of the bone in cases of mild infection even without treatment, but usually early drainage is necessary for the preservation of the bone and periosteum. Where the infective process is confined to the periosteum, simple incision is sufficient, but it is not always possible to determine in early cases whether or not the medulla has been infected, and where doubt exists a small opening should be made into the medullary cavity, for the purpose of relieving tension.

2. **Sequestrum formation** furnishes one of the most interesting and at the same time one of the most unmanageable features of acute osteomyelitis of the jaw, because of the tendency of these bodies to retard complete bone regeneration and to furnish a culture medium for attenuated microörganisms, which after long periods of time may light up renewed infections in the same bone or fresh inflammation in others.

The irritation produced by the long-continued presence of a sequestrum may cause a hyperostosis or sclerosis of the surrounding bone. Removal of the sequestrum is always necessary for complete repair of the bone. Occasionally, it is extruded spontaneously through the soft tissues, but more commonly it must be removed by surgical measures because of the surrounding involucrum.

3. **Metastatic Abscess Formations and Pyemia.**—Pyemia may be the cause or result of acute infectious osteomyelitis and is due to the arrest of the septic emboli not only in the bones but in the soft tissues. Acute pulmonary diseases, as pneumonia and pleurisy, are frequently observed as accompaniments of acute osteomyelitis (Trendel).

4. **Subsidence of the acute symptoms** followed by chronic suppuration.

5. **Joint affections,** as septic arthritis of the temporomaxillary joint, may occur.

Differential Diagnosis.—(1) **Acute articular rheumatism** because of its more common occurrence is the most important condition to be differentiated. However, because of its rare involvement of the temporo-maxillary joint and by its characteristic successive involvement of the larger joints, it is readily recognized.

2. **Tuberculosis of the jaw** comes on slowly, four to six weeks following injury. Tuberculosis elsewhere may exist, especially in the glandular form in children.

3. **Syphilis of bones** which is characterized by its slow onset, chronicity, frequently painless swelling, history of acquired or inherited infection, and the cutaneous, glandular, mucous, and visceral evidence of the disease elsewhere, and its response to appropriate medication.

Treatment.—This is always surgical and consists of relieving the tension and evacuation of the infective material as soon as the diagnosis has been made. The opening should be large enough to provide free drainage and should be preferably located upon the under side of the jaw in order that deformity may be avoided. All carious teeth which might have a causative relationship to the osteomyelitis should be removed.

X-ray examination will reveal the presence of sequestrum or foreign bodies which must be removed before recovery can be expected. In some cases subperiosteal resection of the ramus or body of the jaw is necessary. In these cases it is necessary in order to prevent deformity to bridge over the gap by an autogenous transplant. Dental surgeons are extremely skillful in providing permanent devices in the shape of vulcanite plates and other mechanical means for bridging over the osseous defects which cannot be filled up by autoplastic operations.

Hypertrophied tonsils and adenoids, dermatological lesions, chronic catarrh, and tuberculous glands of the neck should all receive careful treatment as being possible causative factors.

The treatment of tuberculous osteomyelitis is essentially the same as that of the acute type except that one is justified in waiting for the line of demarcation to appear between the necrotic and healthy parts before resecting the bone.

ACTINOMYCOSIS

Definition.—A specific disease of man and cattle due to the streptothrix actinomyces, characterized pathologically by granulomata containing the peculiar sulphur bodies.

Etiology.—The atrium of infection and the primary lesion are usually found in the oral cavity. Various types of streptothrix are known, the bovine differing somewhat from the human variety. In animals the affection is known as big-jaws or lumpy-jaw. The disease is acquired by eating infected grain, especially barley and the commonest lesions are located in and about the mouth. The ray fungi may find lodgment in a carious tooth or lesion of the soft parts, as hypertrophied tonsils, hence the frequency of enlargement of the cervical glands.

Cases have also been reported of inoculation of the lungs following inhalation of dust laden with the ray fungi. It has been stated that the disease may be caused by a number of organisms belonging to this group.

Different strains of the streptothrix are reported to vary as to their growth in the presence or absence of oxygen and their resistance to drying is marked having the vitality to remain alive months under unfavorable conditions.

Actinomycosis of the skin may be secondary to pulmonary or osseus infections with the ray fungus and may closely simulate sarcoma when primary in the viscera. The period of incubation is about four weeks. Children are rarely affected.

Pathology.—Small masses are formed by the development of the ray fungus, which are not unlike tubercles. The streptothrix proliferates in a more or less radial figure, the characteristic rod-like organisms being frequently clubbed at the periphery, resembling spores, the center being occupied by masses of ray fungi surrounded by giant and epithelioid cells. The mass enlarges and after a time breaks down in the center, discharging pus in which the characteristic sulphur grains or iodoform crystals appear. Growths from the pus may be developed on most culture media.

The irritation from the lesion produces an unusually active connective tissue proliferation in the surrounding tissues, amounting almost to a neoplasm. While the tendency of the infection is to spread, the process is a very slow one. The discharging sinuses may heal up temporarily with great heaping up of granulation tissue; later, owing to the accumulation of pus, the sinuses are again opened. This process may be repeated a number of times inducing a great overgrowth of scar tissue with resulting deformity.

Because of the primary lesion being most commonly located in the mouth, the glands at the angle of the jaw are involved early giving the characteristic lumpy-jaw. Pyemia may result from the invasion of a vein. Actinomycosis of the lungs and central nervous system also occurs.

Symptoms.—These are due to the local condition and to the absorption of the products of infection. Mixed infection with the ordinary pus producing organisms frequently occurs. As in other chronic granulomata, pain is not marked unless secondary infection with abscess formation occurs. There is but slight constitutional disturbance except in the visceral forms. The cutaneous lesions resemble sarcoma and are composed at first of firm, painless, bluish or black nodules varying from minute points up to the size of a pea. Later the central area of the nodule becomes necrotic and takes on a pale color, liquefaction takes place and a discharge of pus with sinus formation occurs. Several lesions may fuse and there is extensive heaping up of granulations with widespread induration of the surrounding tissue. Small areas may heal over but the tendency is to ulcerate and invade fresh structures. Spontaneous healing rarely occurs.

Treatment.—Medical treatment consists in administration of large doses of potassium iodid, painting with tincture of iodin, and general supportive measures. Surgically, all infected tissue should be removed and the sinuses laid open and cauterized. No specific treatment is known.

SURGICAL EMPHYSEMA OF THE FACE

Etiology.—This remarkable condition is due to the accumulation of air in the subcutaneous cellular tissue where it has escaped from the air passages through some form of artificial passage. It may follow fracture of the nasal or lacrimal bones or superior maxilla, or penetrating wounds or compound fractures of the frontal sinus. It is observed after cut-throat or stab or bullet wounds of the neck, injuring the air passages or the apex of the pleura. A curious feature of the influenza epidemic of 1918 was the occurrence of emphysema of the neck and face following a fit of coughing. In none of the cases seen was it necessary to provide an escape for the air in the subcutaneous tissues by making incisions over the affected parts.

Pathology.—Extensive surgical emphysema may extend from the tissues of the chest wall following penetrating injuries of both layers of the pleura, the air creeping upward through the cellular tissue of the neck until the face is reached. Penetrating fractures of the ribs may produce emphysema in cases where the wound does not communicate with the outside air. Such a patient produces a remarkable appearance. He is frequently cyanotic as a result of respiratory embarrassment, the normal anatomic landmarks and facial expression are obliterated by the accumulation of air and upon palpation of the inflated area a parchment-like crackling sensation is imparted to the fingers.

In the extreme cases the patient presents the symptoms of asphyxiation and unless relieved, dies of suffocation.

Diagnosis.—Confusion with other swellings about the head and neck is rare because of the characteristic crackling of the skin upon palpation. Infections with the Bacillus Aerogenes Capsulatus while accompanied by the development and accumulation of air in the tissues are so over-shadowed by the element of toxemia that the emphysema is secondary. Cellulitis and erysipelas present a fiery inflammation of the skin in marked contrast to the cyanosis of emphysema.

Treatment.—Consists in the relief of tension by suitable incisions providing escape for the accumulated air. Where possible, the avenue of escape of air from the lungs or air passages should be closed by suture or packed off with gauze.

FROST-BITE

Frost-bite is gangrene or death of a portion of the tissues due to exposure to cold. Susceptibility varies with age, being less likely to occur in full-blooded, active adults than in children and aged persons.

Diseases of the heart and arteries frequently predispose to frost-bite. Debilitated states resulting in anemia and sluggish circulation, as marasmus, tuberculosis and scurvy, are predisposing conditions.

Frost-bite is most commonly observed upon the exposed parts, as upon the ears and tip of the nose. The primary effect of the application of cold to the tissues is vasoconstriction. This accounts for the white, bloodless appearance of the part just frozen. The affected area becomes pale, ischemic, and without sensation. Thrombosis of the vessels occurs and the white blood corpuscles are disintegrated with a liberation of the fibrin ferment. There is a coagulation of the blood serum and this can be demonstrated experimentally by the exposure of a frog's foot to varying degrees of cold. Blood serum has a rather high freezing point. The whole process finally results in dry gangrene and the changes incident to this process are identical with this variety of gangrene due to other causes. In those situations in which only a small area undergoes gangrene, restitution is possible without loss of substance. However, even small areas may be completely destroyed where the freezing has been kept up for some time. When gangrene occurs it is usually of the dry type. The tissues become black and there is little odor to the dead part. In those instances in which moist gangrene occurs there are always present blebs and vesicles, the contents of which become infected with saprophytic organisms. Absorption from areas of moist gangrene is rapid, whereas the intoxication resulting from dry gangrene is frequently negligible. When restitution of the tissues does not take place, a line of demarcation is formed between the living and dead parts, which is evidenced by a hyperemic margin in the healthy tissues. The gangrenous portion is usually thrown off spontaneously and healing occurs by granulation unless infection takes place. In those instances in which inflammation occurs, the changes in the tissues are similar to those of any normal tissue.

Moist gangrene frequently results from too rapid thawing out of the frozen position, leading to the formation of blebs and vesicles which almost always become infected. Superficially, frost bites likewise are more likely to result in moist gangrene, while more extensive or prolonged freezing of the part produces dry gangrene because of the thrombosis of the vessels. There is always a certain amount of inflammation and infection of the surrounding living tissue and this may terminate when the gangrenous portion is sloughed off by a process of necrosis, or it may go on to a spreading infection. These results depend more or less upon the resisting powers of the individual.

When death of the tissue does not follow frost-bite there is a marked hyperemia resulting from the sudden opening up of the vessels in the process of thawing. This hyperemia is accompanied by all sensations ranging from tingling to severe pain according to the degree of the frost-bite and symptoms in the area involved may persist for a long time after the appearance of the affected part becomes normal.

One attack of frost-bite devitalizes the tissues and predisposes to subsequent involvement upon exposure to cold.

Treatment.—When complete death of the part from gangrene has not occurred, the involved area should be vigorously rubbed with snow or ice. Immersion of the part in cold water accompanied by massage and friction until bubbles begin to rise should be tried when no reaction follows friction with snow or ice. Attempts at restitution of the circulation should be maintained for some time even though at first apparently unsuccessful. When signs of returning vitality appear, as indicated by return of warmth and color to the part, gentle massage or dry friction should be instituted and the snow or ice dispensed with. Later the part should be wrapped in some soft warm substance, such as absorbent cotton, in order to preserve the warmth and thereby produce a vasodilatation, encouraging the flow of blood through the compromised vessels. It is always unwise to bring persons who have been subjected to severe low temperatures or moderately low temperatures for a long time suddenly into a warm atmosphere. It has been reported that soldiers, especially in the Russian army, have collapsed upon being brought into a warm room after having stood prolonged exposure in low temperatures.

When the line of demarcation involves bones of ligaments, it is frequently necessary to assist the natural process of gangrenous ulceration by amputation of the bone or division of the ligaments and tendons. The greatest care should be exercised to prevent infection in these tissues which have been devitalized. In the case of moist gangrene with spreading infection it may at times be necessary to do an amputation above the infected area without waiting for a line of demarcation. However, these cases are extremely rare, as in most instances it will be found wiser to wait for the line of demarcation to form.

While paying attention to the local lesion, the patient's general condition should not be overlooked. Hot stimulating drinks and warmth to the body should be given and if possible, the patient put to bed where an even temperature may be maintained. Intravenous hot saline injections or hot enemata may be tried. The use of alcohol is of doubtful help.

BURNS OF THE FACE

These are especially common in children and may be divided into three degrees, as in other situations of the body.

Burns of the first degree consist of a simple erythema or dermatitis, resulting from slight exposure to heat. This is best illustrated by ordinary sunburn or chapped face consequent upon prolonged exposure to sun or wind. In this type no incineration of the tissues occurs, but after a few days the epidermis may be peeled off in strips. There is an area of local hyperemia lasting from one to two weeks, but no scar remains. During this time there may be in the first few days following exposure itching and burning, but rarely any severe pain.

In burns of the second degree vesiculation and blistering result from the more prolonged exposure to heat. The epidermis is destroyed and oozing of serum takes place. The hair of the head, eyebrows, and eyelashes may be lost, but readily grows out again because the deeper layers of the epidermis which contain the hair follicles are not destroyed. Frequently, there are islands of skin remaining intact, from which the epidermis grows until the denuded area is covered. Scarring rarely occurs in burns of the second degree.

In burns of the third degree charring or complete destruction of the involved tissue results. The corium and deeper structures of the skin are destroyed, so that when healing takes place, it must always be by scar formation.

Burns of the head are usually not extensive or deep when the patient lives, because inhalation of the flames necessarily occurs when there is prolonged contact and this causes practically instant death. In those with thick hair the scalp is protected from deep burns by means of the hair. Usually the most extensive burns of the face are caused by explosions or electric currents or flashes, causing instantaneous exposure to a high degree of heat. Burns may be produced by radiated heat, by flames from liquids or solids, or by the *x*-ray or radium.

Pathology.—First-degree burns only produce hyperemia. At times there may be some epithelial desquamation, but the process clears up rapidly without cicatrization as soon as the irritating source is removed. However, repeated burns of the first degree, as observed in sailors or those especially exposed to the wind and sun, may result in malignant degeneration. Single extensive burns of the first degree may produce fatal shock. This is to be expected where more than one-half of the skin is involved.

Second-degree burns rarely produce scarring. In this type of burns large blebs and vesicles form which at first contain clear serum. This later is transformed into pus when a secondary infection of the contents of the blebs and vesicles with a pus-producing organism occurs. Pressure upon first and second degree burns produces an ischemia, while pressure upon third-degree burns produces no change in color, as the blood-vessels supplying the area are either thrombosed or destroyed. When left untreated, burns of the second degree usually become infected and the whole involved area becomes intensely foul and gives off a thick purulent discharge. The blebs and vesicles rupture spontaneously or are opened aseptically by puncture with knife or needle. The serum or pus discharged over the burned area unless removed by irrigation, rapidly dries and forms a crust, protecting the underlying raw surfaces from painful contact with the air or with surrounding objects. Healing occurs beneath the spread of dessicated serum and necrotic plaques. This is finally separated *en masse* from the surrounding healthy area and shed as a more or less extensive sheet, leaving a clean granulating or healed surface.

Because of the rapid absorption resulting from accumulation of septic material upon this raw surface, the symptomatic manifestations are very great, frequently resulting in high fever, scanty, albuminous urine and great prostration with delirium or coma. Second-degree burns about the head are frequently accompanied by acute conjunctivitis, which often becomes suppurative.

Burns of the third degree comprise those in which there is destruction of the vitality of the true skin up to complete incineration of the part. The appearance of the burned area varies with the type of heat. Burns caused by live steam leave the skin pale and bloodless. The actual flame or radiated heat produces a grayish-brown or back color. *X*-ray and radium burns frequently become pigmented. The involved area may be dry or moist. Duodenal ulcer or acute nephritis may follow extensive burns of any degree.

Third-degree burns of the face or head are practically always fatal in children because of the frequency with which the flames are inhaled. In adults they are among the worst of all forms of injury, because of the deep destruction of tissue and the resulting scar formation, causing the most hideous types of facial deformity by contracting scars. This often happens in spite of the most careful treatment.

Symptoms.—The symptoms of burns may vary from a mild irritation, the result of a dermatitis, to profound shock and sudden death. In burns of moderate severity the patient has intense local pain, because the sensory nerve endings in the skin are laid bare. There is frequently a sharp rise in temperature because of the rapid absorption of the toxic material from the large denuded surface. Leukocytosis is present. The urine beomes scanty in amount and shows albumin and casts. Pain is the dominant symptom and in most cases requires morphine for its control. The gravity of the patient's condition depends rather upon the extent than the degree of the burn. Burns about the head are frequently followed by shock. The occurrence of chills following a burn is significant of the flooding of the circulation with the products of infection and is always to be regarded as an unfavorable symptom. Successive chills indicate renewed absorption. Elevation of temperature is present during the chills and may be accompanied by nausea and vomiting and diarrhea, which at times is bloody. Septicemia and pyemia are late complications. Delirium tremens commonly supervenes in alcoholics. In children bronchopneumonia frequently follows extensive burns about the face and head.

Treatment.—The open form of treatment is best suited to most burns of the face. In order to prevent the pain resulting from contact with drafts of air, it is best to put the patient in bed and make a framework extending from the head to the foot of the bed over which the bed clothes can be thrown in such a manner that currents of air cannot strike the burned surface. The denuded area should be carefully washed off by a stream of sterile water or salt solution. This should be done at least once a day or oftener if there is any accumulation of pus.

Vesicles and bullæ should be opened with the greatest aseptic precautions and syringed out with normal salt solution. The application of carron oil or soda bicarbonate solution by means of gauze bandages has been used for many years, but each dressing is extremely painful, the granulations growing up into the meshes of the gauze being torn off each time the dressing is removed leaving a bleeding surface with exposed nerve endings. This constant irritation leads to an overgrowth of connective tissue and thick contracting scars. Paraffin has recently received a great deal of attention in the treatment of burns of the second and third degrees. Its chief virtue lies in the fact that it does not adhere to the underlying tissues and may be removed without tearing off the granulations. Furthermore, it produces a smooth, soft covering to the denuded area and protects it completely from air currents and bacterial contamination. It is not best suited to burns of the face. However, it may be very satisfactorily used about the neck and it is in this situation that some of the worst deformities occur.

In order to prevent contraction of the scar tissue in those situations where there has been a deep destruction of the tissues, it is a good plan to put on some form of orthopedic apparatus to keep the part in extension. For example: In burns of the anterior surface of the neck and lower jaw a plaster of paris collar 4 or 5 in. in width will keep the chin elevated and all the tissues stretched to their fullest extent, and in most cases will prevent a great deformity. In many instances no form of dressing or treatment will prevent contracting scars and it is in these cases that plastic operations for the purpose of covering the denuded area with healthy skin must be resorted to. The most favorable situations from which to take pedicle grafts for the neck or face are from the arm or forearm. The pedicle should be left intact for a week or 10 days and then cut. It is wise to have the base of the pedicle as wide as possible and to construct the flap in such a manner that it may not be twisted at its base, thereby hazarding its nutrition. The denuded area from which the flap has been removed may be covered by Thiersch grafts, the skin preferably taken from the anterior suface of the thigh.

Systemic Treatment.—Opium in some form is practically always necessary for relief of the pain in the first 48 or 72 hours following burns. In case of shock intravenous normal saline solution to which has been added adrenalin in amounts to be governed by the patient's age and condition of the bloodvessels is very helpful. It is important to keep up elimination at the highest possible level, because of the rapid and continued absorption of the toxins from the burned area. A light nutritious diet should be maintained and the patient's strength conserved in every possible way.

X-RAY BURNS

X-ray burns may be of three degrees, as are burns of ordinary origin. The acute lesions of *x*-ray burns more commonly occur in patients subjected to intensive radiation, while the chronic affections are restricted to operators and those more or less constantly exposed to the influence of the rays.

X-ray burns of the first degree are usually not felt by the patient for 10 to 20 days after exposure. The process begins in the true skin and not upon the epidermis. Simultaneously with the smarting and pain there is pigmentation of the area. The cutaneous appendages as hair and eyebrows are shed but are not permanently lost. The superficial epidermis peels off as in ordinary sunburn, leaving a smooth sensitive hyperemic surface. This usually heals of itself, but frequently requires the application of soothing and protective ointments to allay the pain and itching.

X-ray burns of the second degree result from more prolonged exposure to the rays and require a longer time for development than do first degree burns. The formation of bullæ and vesicles may be preceded by dermatitis as in the first variety. Edema of the part frequently occurs and upon puncturing the bullæ and wheals there is considerable discharge of serum which does not tend to become inspissated and form crusts, as in burns of the usual type. In the absence of the protecting crusts commonly observed, the denuded areas are exquisitely sensitive to touch or draughts of air. This type tends to heal without loss of tissue if the wound is kept clean, but the vitality of the tissues has been lowered and healing takes place slowly.

X-rays burns of the third degree follow intense or oft repeated exposure. Gangrene of the true skin ensues leaving areas of chronic ulceration which show no tendency to heal, but on the contrary frequently degenerate into malignant growths. The affected area at first becomes dead white, hence the term "white gangrene" as applied by G. G. Hopkins (Da Costa). Later ulceration takes place and sloughing of the involved tissues begins. Secondary infection with pus-producing organisms occurs in third-degree *x*-ray burns as in those of the ordinary type. There is little effort at repair and the denuded area becomes surrounded by a border of brawny infiltrated tissue. The stimulation produced by the irritating discharges causes an excessive hyperplasia of cells which frequently take on the characteristics of malignancy.

Treatment of third-degree *x*-ray burns is notoriously discouraging. All sources of irritation must be removed and the denuded area rendered as nearly aseptic as possible. Skin grafting by the Thiersch method or by the pedicle flap will frequently heal the ulcer. Failing in this, wide excision of the affected parts including the floor of the ulcer followed by a suitable plastic operation is necessary. When occurring in the fingers and hands amputation is often necessary.

NEVUS

(Nevus Simplex; Portwine Stain; Claret Stain; Birthmark; Feurmal; Tache de Feu; Angioma and Angioma Simplex)

Etiology.—Although this lesion is congenital, it frequently not only enlarges greatly in area after birth but may also change histologically, taking on the character of an angioma, cavernous angioma or even a cirsoid aneurysm. It may vary in size from a scarcely perceptible stain to a large area, raised above the surrounding skin.

Harvey Cushing calls attention to the fact that Bärensprung, Etienne and others have emphasized the fact that the development of nevi often confines itself to the cutaneous area supplied by one or more of the sensory branches of the trigeminus nerve. This strongly indicates a distubance of the sensory nerves or ganglia (Cushing). The common occurrence of spastic hemiplegia in children with nevi corresponding to trigeminal branch distribution is a further proof of a neuropathic etiology. Nevi might properly be included with Herpes Zoster in the classification of Vascular diseases of nervous origin.

Histology and Pathology.—These vascular lesions occur as capillary telangiectases producing varying degrees of discoloration of the skin from the bright-red of arterial blood to the dark-wine color of venous blood. They are usually found upon one side of the face or scalp but may cover large areas upon the body. Upon palpation no elevation of the discolored area may be noticed, but as the condition develops and vascular channels are formed they may be felt as smooth elevations or nodules. When sacculation of the vessels takes place the tumor becomes plum colored from the venous stasis.

Symptoms.—Usually no subjective symptoms are noted unless the lesion assumes large proportions and the vessels take on the character of a cirsoid aneurysm, in which event the patient may become conscious of pulsation and experience attacks of vertigo upon sudden changes of position or upon physical exertion. Too little attention has been given to the influence of such an unsightly facial blemish upon the development of the character and disposition of the child. Children handicapped by the presence of nevi upon the face or head should be given every moral support and efforts should be made to combat the diffidence which is likely to develop. Frequently, these children dread the meeting of strangers or appearance in public places, as schools or social gatherings, resulting in that lack of normal mental growth which can only take place under the stimulation of active participation in the work and sports of other children.

Diagnosis and Prognosis.—The congenital origin of nevi together with their characteristic appearance either as small portwine stains in their simpler forms through all the stages of their development up to the complex cirsoid aneurysm make their recognition easy.

The prognosis as to life is good, although severe hemorrhage may take place in growing capillary nevi in infants. Stellwagon mentions a case of repeated hemorrhage from a capillary nevus of the vessels in the ear in which ligation of the larger superficial vessels gave no relief until the main artery was ligated.

Spontaneous retrogression and even cure in capillary nevi have been noted. This has at times been attributed to a mild local trauma. The ulceration attendant upon local atrophy of the skin may sometimes cause hemorrhage or infection. The prospect for cure is much better in the small capillary nevi than in the elevated portwine stains or cavernous lesions.

Treatment.—The indication for treatment in the simple angiomatous type is clear and this should be undertaken at an early date.

Carbon dioxide snow and radium and x-ray have given excellent results in the treatment of the simple form. Pressure exerted by collodion dressings combined with puncture either by an ordinary steel needle or in combination the electric current has been used with good results. This procedure frequently must be repeated a number of times. In the larger firmly established growths excision by the knife or galvo-cautery is necessary. This necessitates grafting of the denuded area by the Thiersch or flap method and is frequently a formidable operation because of the great difficulty in controlling hemorrhage. Malignant degeneration has occurred in those forms of nevi which break down and ulcerate.

LIPOMA

These tumors are masses of fatty tissue usually acquired, although congenital lipomata have been observed, situated in the skin or subcutaneous tissue and composed of a connective tissue reticulum supporting fat lobules. They resemble ordinary adipose tissue and are more commonly observed upon the back of the neck, back and buttocks, but may occur anywhere. In the liver and kidneys they can be detected only by microscopic examination, while the pedunculated masses of the neck and back frequently weigh several pounds. Except when occurring within the joints or in the arborescent form, lipomata always have capsules which demarcate their structure sharply from surrounding tissues.

Their development is usually slow and painless, and symptoms arise only from pressure or mehanical interference. While the occurrence of a lipoma is usually solitary, symmetrical or multiple growths occur.

Infection with abscess formation may take place. Mucoid changes may occur, leading to the development of a myxolipoma.

Their treatment consists in radical removal Usually this can be satisfactorily done under local anesthesia, and is to be preferred except in the arborescent form which is frequently widely disseminated in the surrounding tissues and is very difficult to remove.

DERMOID CYSTS

These tumors are usually congenital and of slow growth. They develop in special locations and are frequently adherent to the bone and upon removal reveal a depression or a complete defect of the underlying osseous tissue. In the orbit the dermoid is situated usually at the outer or inner canthus.

The tumors are lined by skin and contain the usual cutaneous elements, such as hair and sebaceous and sweat glands. The cyst may be filled with sebaceous material. The so-called sequestration dermoid develops from the remains of embryonic epithelial elements especially in the region of the orbit or the scalp. In those cases where a bony defect exists the cyst may be connected to the soft tisses upon the under side of the bone by a pedicle. This is especially true in the scalp where they may be confused with meningoceles.

Dermoids appear as rounded smooth tumors rarely reaching the size of a walnut, of soft consistency and painless. They are not attached to the overlying skin.

They also occur in the nasofacial sulcus, on the bridge and at the tip of the nose, and doubtless develop from the remains of fetal epithelium which has been enfolded or sequestered. Along the bridge and tip of the nose dermoids develop as a consequence of incomplete closure of the median sulcus and persist as narrow defects lined by skin from which develop epithelial structures. Hairs occasionally protrude from these narrow defects.

In the scalp the most common situations of dermoids are over the anterior fontanelle and over the occipital protuberance. These cysts likewise result from the grafting or enclosure of embryonic epithelial elements or rests in the subcutaneous connective tissue. A curious fact in connection with the development of dermoids of the scalp is the connection of the tumor with the underlying dura mater by means of a fibrous pedicle, frequently causing the growth to resemble a meningocele. In cases where no natural opening existed in the skull the pedicle was found to have passed through a defect in the atrophic skull (Bland-Sutton). These tumors may reach a larger size than those of the orbit or nose and often pulsate. They expand and collapse with respiratory efforts, such as coughing and sneezing, thereby further simulating a meningocele.

The treatment consists in the removal of the cyst wall where possible. When this cannot be done, the lining epithelium should be destroyed by the cautery after evacuating the cyst contents. When a pedicle exists as much of this should be removed as possible.

IMPLANTATION CYSTS

These cysts are also called traumatic or acquired dermoids and arise from the carrying into the deeper structures of portions of skin containing sebaceous and sweat glands. They occur most commonly upon the exposed surfaces of the body as the face and hands, but may occur anywhere upon the skin. Punctured wounds caused by small penetrating objects as nails, thorns, slivers, etc., are chiefly responsible, although traumatic dermoids have been observed following contused and lacerated wounds. Theoretically small pieces of skin are carried down into the deeper tissues by the penetrating force and implanted in the depth of the wound. Under favorable conditions the graft takes and a structure resembling skin develops. Later the more complex structures such as hair and sebaceous and sweat glands appear.

These implantation dermoids rarely grow to exceed the size of a marble. They are smooth, oval or round, painless, and frequently fluctuation may be made out. Infiltration of the surrounding tissues does not occur and none save mechanical symptoms are produced.

Treatment consists in the enucleation of the sac and where that is found to be impossible, curettement of the contents and destruction of the lining epithelium by the cautery.

THYROGLOSSAL CYSTS

"There are several embryonic canals which normally are obliterated in development, *viz.*, the thyro-glossal duct, the post-anal duct, the omphalo-mesenteric duct, and lastly branchial clefts" (Bland-Sutton).

The thyro-glossal duct is a tubular prolongation from the ventral surface of the pharynx which divides about the level of the cricoid cartilage, the upper portion going to the tongue and the lower to the thyroid isthmus. Bland-Sutton says the isthmus of the thyroid in fetal life may be considered as a secreting gland with duct emptying into the pharynx. The foramen cecum represents the end of the upper part of the thyro-glossal duct which may be partially or completely obliterated. When the upper end is closed and the rest of the duct remains open a retention cyst results, the contents being composed of desquamated epithelium, sebaceous material and occasionally epithelial structures. The cyst wall is fibrous and it is lined by epithelium. The mass may be present at birth but more often develops at a later time.

Treatment consists in the removal of the cyst wall and contents.

MEDIAN CERVICAL FISTULAS

These arise as a result of the obliteration of a portion of the thyroglossal duct and according to Bland-Sutton are never congenital but appear several years after birth as a median swelling in the neck below the hyoid bone. In some instances a discharge of mucus may be observed before the cyst is opened. Upon opening the intact sac a discharge of mucus occurs. If a probe be introduced it will enter in an upward direction in the median line for a variable degree. The fistula is lined by epithelium and may contain hair or other epithelial structures.

The treatment consists in the removal or destruction of the lining epithelium.

ACCESSORY THYROIDS

As the isthmus of the thyroid takes its origin from the embryonal thyroglossal duct it is probable that anomalies in the course or development of this duct might cause the implantation of thyroid elements in anomalous situations. Accessory thyroids may be found in the neighborhood of the isthmus or laterally as far as the anterior margin of the sternomastoid muscle. When they remain quiescent, they give no trouble but strangely seem to partake in the pathological changes of the main gland, becoming much larger in cases of goiter.

Treatment consists in the removal of the enlarged accessory thyroids with the main goiter.

BRANCHIAL CYSTS

The human embryo contains four branchial clefts upon each side. The upper corresponds to the external auditory meatus and becomes the Eustachian tube, while the remaining three are situated approximately along the anterior border of the sternomastoid muscle equidistant from each other, the lowest one being just above the sternal insertion of the muscle.

Normally these clefts become closed before birth, but occasionally obliteration fails to take place and the result is an open canal reaching for a variable distance upward and communicating in some instances with the pharynx. Attention may first be directed to these openings by the presence of tags of skin situated at the mouths of the canals and known as cervical auricles.

The clefts are lined by mucosa which secrete a variable amount of fluid.

In the process of obliteration of the canal one or both ends may remain open, together with the persistence of a variable distance of the lumen of the canal. The pockets thus formed may become filled with mucus or sebaceous material. Instances of the finding of epithelial structures in these cysts have been reported. Infection of the lining membrane and contents may occur, producing an abscess. Pockets with internal orifices, e.g. communicating with the pharynx have been diagnosed as esophageal diverticula. The treatment consists in the removal or complete destruction of the lining membrane.

ERYSIPELAS

Erysipelas is an acute contagious disease caused by the streptococcus erysipelatis and characterized by blocking of the smaller lymphatics of the skin by the pathogenic flora and infiltration of the subcutaneous cellular tissue. The mucous membrane may also be attacked.

Etiology.—Streptococcus erysipelatis is found in the smaller lymphatics of the skin and in the cellular tissue. There is reason to believe that this organism is identical with the streptococcus pyogenes but of a different strain. Many authorities believe that other pyogenic organisms in attenuated forms may cause erysipelas.

The infection gains entrance by way of wounds of the surface in the form of lacerations, punctures or abrasions. More frequently, however, there is no discoverable lesion of the skin or mucous membrane, constituting the so-called idiopathic infection.

The disease occurs frequently in persons of low vitality brought on through lack of proper nutrition, of improper living conditions and the use of alcohol or drugs. It is frequently observed in the course of nephritis and diabetes. Epidemics have been observed in hospitals and in asylums, which is an argument in favor of the isolation of the cases when the diagnosis of erysipelas has been made.

Pathology.—The infecting organisms are found in areas surrounding the classic elevated red margin. These cocci are found in chains and many of their cultural peculiarities are identical with the streptococcus pyogenes. They are also found in the wall of active inflammation beneath the raised margin. In this situation the lymphatics are blocked by the rapid proliferation of the pyogenic flora and there is frequently a focus of lymphangitis or perilymphangitis. The regional lymph glands are frequently enlarged and painful and the lymphatic channels draining the erysipelatous area may frequently be palpated as red indurated cords radiating to the nearest filter glands. A persistent enlargement of these lymphatic glands may take place after the subsidence of the erysipelatous infection.

When the infection gains entrance through macroscopic lesions of the skin normal wound healing may apparently proceed for four or five days. The wound edges then take on a dirty yellowish-brown appearance and the edges of the wound separate, exposing the deeper structures. Secondary infection very commonly occurs with the formation of pus and great tissue destruction. The surrounding infected area takes on a rosy-red color, feels smooth and tense to the palpating fingers and is demarcated from the surrounding healthy tissue by a slightly elevated irregular margin. In those situations in which the subcutaneous connective tissue is very lax, as in the eyelids and scrotum, there is a brawny induration. The tension becomes great and because of the lymphatic and venous stasis, gangrene of the involved skin may occur. Ordinarily, however, as the eruption fades there is a brawny desquamation of the epithelium, leaving a normal skin with no trace of the former infection.

A pure erysipelatous infection is self limited, the disease running its course in from one to three weeks. When recovery ensues the tendency to subsequent attacks is very much increased.

Symptoms.—The onset of symptoms may be preceded by headache, and drowsiness for 12 to 24 hours. Following this malaise, chilly sensations or severe rigor may initiate the onset of the disease. In children convulsions frequently precede the onset of the fever. If the temperature be taken during the chill or convulsion, it will be found markedly elevated. The pulse is rapid and a leukocytosis is present. With the onset of the constitutional symptoms the characteristic rose-red rash appears. The fever lasts as long as the cutaneous eruption. When occurring upon the face, the eruption is usually quite symmetrical, spreading laterally from the bridge of the nose in the shape of a butterfly and fading upon pressure. It is rarely painful, thus distinguishing it from cellulitis, but the patient may complain of stiffness of the skin, burning or smarting. Itching is sometimes a disagreeable manifestation. As the eruption spreads, vesicles or bullæ may make their appearance. Upon opening these a clear serum exudes, which in severe cases may be even purulent.

Delirium is common and begins frequently on the second day. In the more severe cases the delirium deepens into coma from which the patient cannot be aroused. In ordinary cases, however, the nervous manifestations subside with the fever and eruption. The duration of an attack is from one to three weeks.

Diagnosis.—Cellulitis is practically the only condition which may cause confusion in the diagnosis. However, in erysipelas the rapidly advancing rose-red rash, limited by the elevated margin of the skin, clearing up in the center and leaving a brownish-yellow desquamated surface, usually without destruction of the tissue, suffices to differentiate the conditions. An infected wound in which there is a collection of pus may at first simulate erysipelas, but the fluctuation, evidences of suppuration, history, and the securing of pus by aspiration clears up the diagnosis. Erythema nodosum does not progress symmetrically as does erysipelas and is unaccompanied by evidences of toxic absorption. More commonly it affects the lower extremities and occurs in rheumatic individuals. Sunburn and acne rosacea are differentiated by the history and by the fact that the inflammation of the skin does not spread.

Prognosis.—This depends almost wholly upon the underlying conditions. In young vigorous persons the prospect of complete recovery is very good. In aged or debilitated erysipelas is frequently a terminal condition. In epidemics in hospitals or asylums the death rate is high.

Erysipelas is usually not so dangerous in itself, but occurring as a complication in pathologic states is frequently fatal. In infants the mortality is high. Coley has made practical application of the clinical observation that chronic wounds and neoplasms have disappeared after erysipelatous infection, by treating sarcomata by various combinations of sterilized cultures of streptococci and other pyogenic organisms injected into the tumor. Unfortunately successful results are not always obtained.

Treatment.—The patient should be isolated as soon as the diagnosis is made. Local applications of solutions of belladonna, opium or ichthyol in 20 to 40 per cent. solution relieve the burning and itching sensations. Scarification of the advancing raised margin with injections of sublimate solution, silver nitrate or iodin have been tried, but without much success. Antistreptococcic serum has been given subcutaneously, but there is no specific therapy. Where great tension of the skin exists incisions are advisable. When the process reaches the neck causing respiratory embarrassment, tracheotomy is sometimes necessary.

Where the erysipelatous infection attacks the tissues of the fauces local applications of silver nitrate in 0.5 per cent. strength should be tried, great care being taken that none of the solution drips from the applicator into the larynx.

Constitutional treatment in many cases is far more important than the local management and consists in aiding elimination and keeping nutrition at the highest level possible.

Erysipelas of the Fauces.—This is an infection of the mucosa of the fauces, sometimes spreading to the glottis and larynx. It is rarely an independent infection, but usually secondary to a focus of erysipelas elsewhere. The tissues of the fauces and palate are edematous and plum colored. Dyspnea may occur because of the edema or spasm of the glottis. The voice is husky or entirely lost. Sloughing in this type of erysipelas is not uncommon. As in most throat infections the amount of toxic absorption is unusually high.

The **treatment** is as for the cutaneous type.

ORTHOPEDIC SURGERY

EDITED BY ARTHUR STEINDLER, M.D.

IOWA CITY, IOWA

CHAPTER CVIII

GENERAL PATHOLOGY OF BONE IN CHILDREN

BY D. B. PHEMISTER, M.D.

CHICAGO, ILL.

PYOGENIC OSTEOMYELITIS

Etiology.—Predisposing Causes.—Pyogenic infection of bones is a disease especially of childhood and adolescence, more than one-half the cases occurring between the ages of 10 and 16. It is comparatively rare during infancy, although many cases are recorded in which the microörganisms gained entrance into the child from an infected nursing mother. It decreases rapidly in frequency as the growing period comes to a close and the cartilaginous epiphyseal lines disappear. Boys are more frequently affected than girls, the proportion being about three to one. This is the result of greater activity and exposure on the part of the male sex. Damp and cold seasons predispose to osteomyelitis. The infection may arise spontaneously in healthy children without any predisposing factors, but frequently there is a history of fatigue or exposure to wet and cold. More commonly a focus of infection exists in some part of the body from which microörganisms gain entrance to the circulation and lodge in the bone. Boils and infected wounds, abrasions and blisters are the commonest causes. Carbuncles, paronychiæ, chronic ulcers and secondarily infected skin diseases are less frequent causes. Bone involvement is more apt to develop early, but it may appear late in the course of the skin infection. Subcutaneous cellulitis or phlegmon may also give rise to osteomyelitis. Acute localized infection in the upper respiratory tract is rarely a precursor of osteomyelitis, as contrasted with arthritis. Acute rhinitis, pharyngitis and tonsillitis are comparatively rarely met with, the latter being the most frequent. Chronic infections of the teeth, tonsils, accessory sinuses and middle ears apparently play an unimportant rôle in the production of the disease.

Osteomyelitis arises occasionally as a complication of infections of the lungs and pleura, especially in pneumonia, lung abscess, bronchiectasis and empyema. It is very rarely a complication of such contagious diseases of children as measles, scarlet fever, whooping-cough and diphtheria. Nearly all cases with a generalized rash and osteomyelitis are not scarlet fever, but staphylococcus pyogenes aureus osteomyelitis with sepsis and a secondary scarlatiniform eruption. Influenza rarely gives rise to osteomyelitis. Bone involvement in typhoid fever arises usually from infection by the typhoid bacillus, but occasionally from a complicating streptococcus or staphylococcus infection. Septic processes of all forms may be complicated by bone infection. Small-pox may give rise to osteomyelitis, and more frequently in children than in adults. It results from the entrance of pus organisms and particularly the staphylococcus through the eruptive lesions of the skin and gives rise to hematogenous infection of the bones. It should not be confused with the variola lesion in the region of the epiphyseal line, which frequently results in destruction of the latter with arrested longitudinal growth. A great many cases of osteomyelitis arise in patients who on examination present no evidence of a focus of infection elsewhere, so that the portal of entry of the microörganism cannot be determined.

The relation of traumatism to hematogenous osteomyelitis is a much disputed point. A history of previous moderate traumatism from falls and blows is very frequently obtained, and this may be a point of importance in the localization of bacteria in the bones. However, a great deal of doubt is created by the fact that severe injuries resulting in closed fractures are very rarely complicated by the development of hematogenous bone infection at the seat of injury. Lexer showed that young animals in which staph. pyog. aur. is intravenously injected after traumatism of the bones may develop infection at the seat of traumatism. Compound fractures are infected as a result of the entrance of bacteria through the wound, producing a localized osteomyelitis, but secondary osteomyelitis in other bones from infected fractures is extremely rare. Bone plates or wires inserted for the fixation of fractures may heal in aseptically but after years osteomyelitis may develop at their seat. No doubt the bacteria in some of these cases gain entrance from the blood stream. Secondary foci in other bones are a common complication of primary hematogenous osteomyelitis, developing early or late after the onset of the original infection.

Exciting Causes.—The exciting cause is a pyogenic organism, which gains entrance through the blood stream, although rarely the intact bone is directly invaded from without, as in the case of bone felon following an infected abrasion of the finger. The bacteria most frequently found are staphylococcus pyogenes aureus, albus and citreus, streptococcus, pneumococcus, typhoid bacillus and colon bacillus. In my experience the staphylococcus group has produced 90 per cent. of the cases of osteomyelitis. The staphylococcus pyogenes aureus is the organism responsible for the great majority of cases. Pasteur first observed that this germ causes both boils and osteomyelitis and designated the latter as bone furuncle. The staphylococcus pyogenes albus ranks next in importance, but does not occur more than one-fifth as frequently as the staphylococcus aureus. The two organisms are occasionally associated in the same infection. The staphylococcus citreus is very rare. The streptococcus pyogenes plays a minor rôle in the production of osteomyelitis when compared with its importance in infections of the joints. It is apt to arise as a result of streptococcus infection in other parts of the body. Pneumococcus osteomyelitis is rare and usually a complication of pneumonia or acute otitis media, although primary bone infection may occur. Anaërobic bone infection is extremely rare, except as a complication in compound fractures.

Pathology.—A profusion of names have been used to designate the different forms of pyogenic infection of bone. But it is best to include them all under the general head of osteomyelitis, regardless of the location or type of infection, as in the great majority of cases the infection begins in the soft tissues of the interior of the bone. Klemm is of the belief that bacterial lodgment always occurs in the bone marrow, due to the lymphatic tissue which it contains. The fact that lymphatic tissue is more abundant here, as in other organs, during youth explains the frequent occurrence of osteomyelitis at that time. This view is not free from objections. While the infection begins in the soft tissues of the bone, it does not always start in the regions of bone marrow richest in lymphatic tissue, but is more prone to begin in the zone of most active growth, especially at the ends of the shaft, where lymphatic tissue is less abundant. The presence of large vascular spaces in the growing regions with resultant sluggish circulation and opportunity for bacterial lodgment is a factor of great importance in the localization of the disease. It has long been considered that the terminal branches of the arteries supplying both the ends of the shaft and the epiphyses are end arteries and that bacterial embolism in these regions played a rôle in the production of osteomyelitis. The recent investigations of Nussbaum have shown that the terminal branches of the metaphyseal arteries supplying the bone of the end of the shaft are end arteries, but those of the epiphyses are not end arteries. This may account to some extent for the frequency of primary infection of the end of the shaft and its extreme rarity in the epiphysis.

The inflammatory reaction is the same in pyogenic infection of bone as in that of the soft parts, but the effect on the tissues is widely different in some respects. This is due to the presence of lime salts in the stroma of the bone, which are not removed by the destructive agents of the inflammation in the same way as are the organic elements. Soft parts that are killed by the inflammation usually undergo rapid disintegration and liquefaction from the action of the invading leukocytes and the proteolytic substances contained in the exudate. But the lime salts are not removed by the action of either of these agents and are only broken down by the direct action of fixed tissue cells. This requires a much longer time for occurrence, consequently dead bone persists in the field longer than dead soft parts, which greatly prolongs the duration of the infection. Also defects resulting from bone destruction are not obliterated as rapidly as are those resulting from death of soft parts, because of the rigidity and inability to collapse of the bony walls and their slight tendency to form new bone.

Portions of Bone Affected.—The following forms of pyogenic infection may be distinguished according to the portion of bone primarily involved. (1) Primary osteomyelitis of the shaft. (2) Primary periostitis. Nearly all of the infections are primary in the diaphysis and involvement of the epiphysis or periosteum occurs secondarily. Primary epiphysitis is not met with, unless Perthes' disease is a pyogenic infection of the femoral head, which seems quite probable from recent bacteriologic and pathologic studies by Kidner, Legg and Phemister.

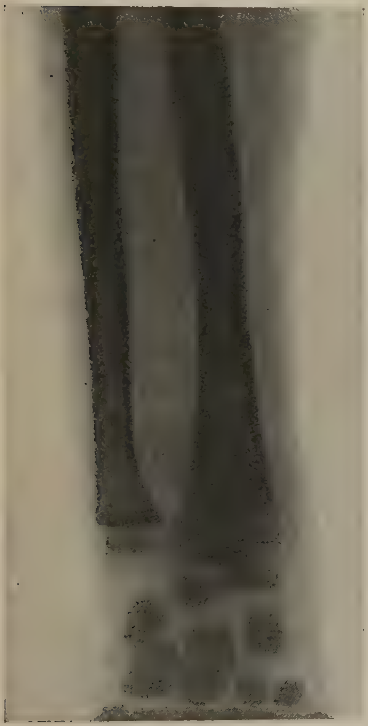

FIG. 1.—Osteomyelitis of lower half of radius of 14 days' duration. Earliest *x*-ray signs. Beginning bone destruction in metaphysis and new bone formation along ulnar side of shaft.

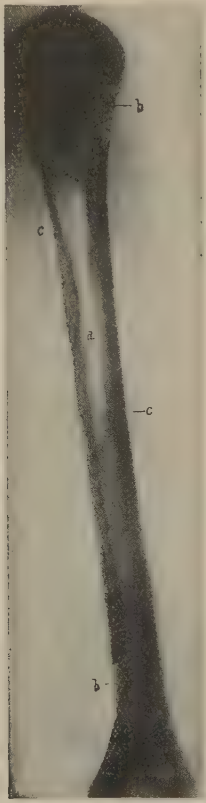

FIG. 2.—Osteomyelitis of humerus, in boy, age 11. Seventeen days' standing. (*a*) Window for drainage. (*bb*) Bone destruction. (*cc*) Beginning involucrum.

Primary Osteomyelitis of the Shaft.—The portion first attacked is usually the metaphysis next to the epiphyseal line, but the disease may start in any portion along the course of the shaft. When the disease starts in the metaphysis, it usually spreads, leading to diffuse infection of a variable amount of the shaft, but when it begins away from the end of the shaft, a localized osteomyelitis is more apt to result.

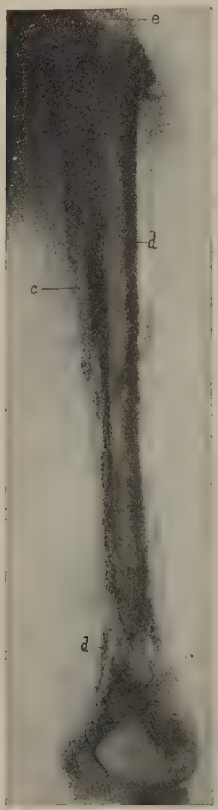

Fig. 3.—Same. Twenty-nine days after Fig. 2. (*c*) Involucrum. (*dd*) Sequestra. (*e*) Pathological fracture.

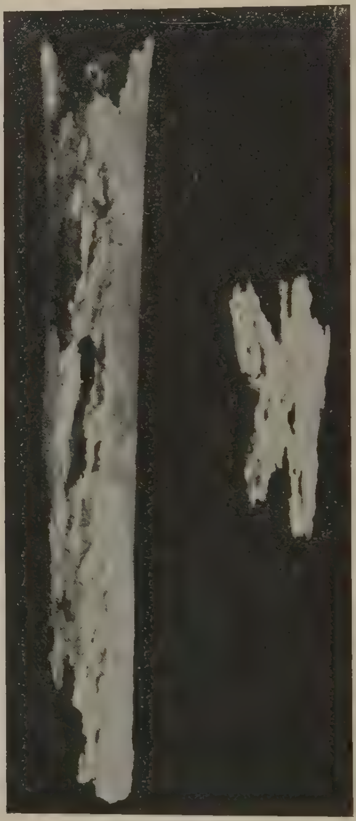

Fig. 4.—Osteomyelitis, sequestra *d*, removed 46 days after Fig. 3.

Diffuse Suppurative Osteomyelitis.—This is the form most commonly seen. The infection begins in the end of the shaft, leading to a diffuse infection of the cancellous tissue. Bacterial toxins are rapidly formed and have a somewhat better opportunity than usual to destroy tissue within the bone because of the anatomical arrangement. The surrounding rigid inexpansible bony wall limits the amount of hyperemia, edema and leukocytic infiltration that can occur, which to some degree diminishes the defensive measures. This may lead to a very rapid necrosis *en masse* of the entire cancellous area. The surrounding cortex prevents the immediate escape of the effusion to the outside, which results in its spread along the medullary cavity to the marrow and cortex away from the primary seat of infection. When seen in the early stage, the necrotic marrow and cancellous bone of the end of the shaft may appear somewhat dry, gelatinous or hemorrhagic without the presence of any considerable amount of pus.

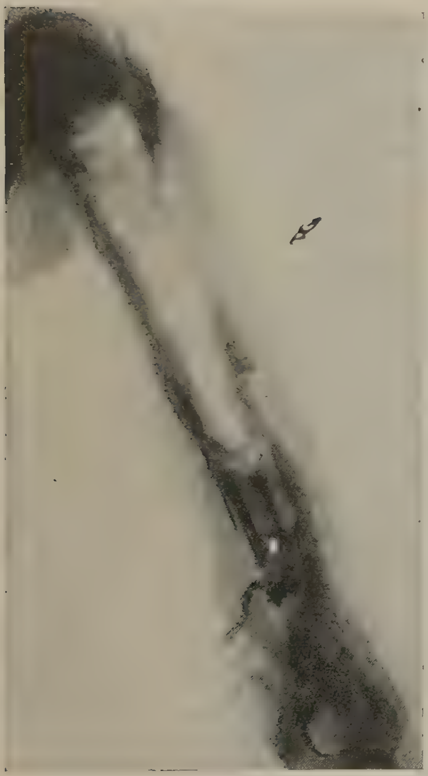

Fig. 5.—Same. Six days after removal of sequestra in Fig. 4.

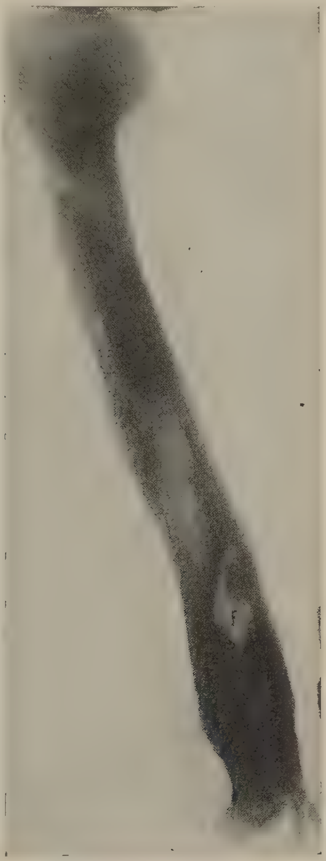

Fig. 6.—Same. Nine months after Fig. 5. (*f*) Cavity causing fistula for six months.

Extension of the inflammation through the cortex first occurs in the metaphyseal regions where it is thinnest and perforated by vascular canals. It results in a subperiosteal accumulation of pus, which separates the periosteum from the underlying necrotic cortex.

Further spread of the infection may occur along periosteum, cortex or endosteum, but involvement of the cortex is dependent largely upon the extent of spread along its surfaces. Necrosis of the entire thickness of cortex nearly always occurs in those regions where both periosteal separation and necrosis of adjacent bone marrow take place. If involvement of the medullary contents extends farther than periosteal separation, there may be absence of or only partial necrosis of the cortex on the endosteal side. On the other hand, if periosteal separation extends farther than endosteal involvement, there may be absence of or varying degrees of cortical necrosis along the periosteal surface.

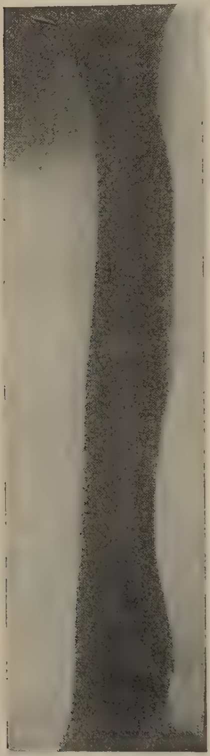

Fig. 7.—Same as Fig. 6. Two years and four months after onset.

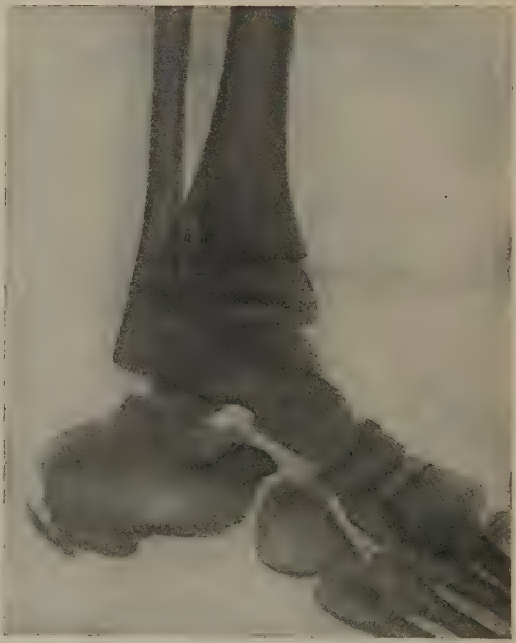

Fig. 8.—Staphylococcus pyogenes aureus abscess (*a*) of six months' standing.

Spread of infection along the medullary canal is of greater importance in the production of necrosis of the entire thickness of cortex in all or portions of its circumference than is sub-periosteal spread. However, abscesses may be found in the medullary canal at considerable distances beyond the limits of dead compact bone and a segment of the cortex may remain unattacked by the infection which spreads through its medullary canal and produces necrosis of the shaft beyond.

Periosteum is always separated from underlying dead cortex, but subperiosteal pus may strip it from surrounding living bone, leading to spread of the infection in this way.

The extent and distribution of bone killed in the first few days of the infection are extremely variable. The dead bone may be confined to a small amount of the cancellous portion at the end, including its overlying thin cortex, or the entire end of the shaft may be involved. In other cases necrosis of the cortex occurs in long irregular streaks or separate pieces for variable distances away from the ends and may extend farther on either the periosteal or the endosteal side. Sometimes one-half or two-thirds of the shaft will be killed in its entire circumference, while in rare cases the entire shaft may become necrotic. There is usually a certain amount of spread of inflammation in the surrounding living cortex without extensive destruction of bone. This gives rise to small, irregularly distributed areas of destruction producing the picture known as rarefying osteitis.

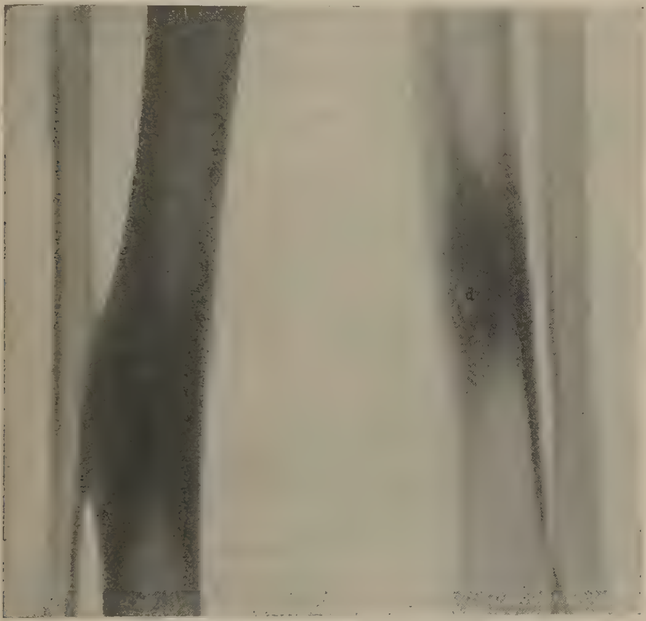

FIG. 9.—Bone abscess of tibia, one year's standing, sixteen year old boy.

Extention of the infection beyond the shaft may occur in a number of ways. The periosteum is usually perforated early and the pus extends into the surrounding soft parts. If not drained surgically, it usually leads eventually to perforation of the skin and fistula formation. Important complications may result from prolonged retention of pus in the soft parts.

Involvement of the epiphysis is an unusual occurrence, as the cartilage offers considerable resistence to the spread of the infection. Extensive destruction of the metaphysis leading to fracture or separation of the epiphyseal line predisposes to infection of the epiphysis.

The commonest lesion is a localized abscess bordering on the epiphyseal line and communicating with the infected metaphysis through an opening in the epiphyseal cartilage. A diffuse suppurative epiphysitis may occur and is more frequent in older children, where the epiphysis is largely ossified and the epiphyseal line narrow. This leads to suppurative arthritis and to more or less necrosis and breaking down of the epiphysis. The cartilaginous epiphyseal line may be partly or completely destroyed, leading to arrested longitudinal growth.

Changes after Spread of the Infection Has Ceased.—The acute destructive changes are most marked during the first week or 10 days, by the end of which time abscess formation in the soft parts had usually occurred and frequently drainage has been established. Then spread of the infection usually abates or ceases, although it may continue to extend for some time, especially if the proper surgical treatment is not carried out. After localization of the infection, reparative changes assume a prominent rôle in the disease. They consist mainly in the formation of granulation tissue and new bone. The granulation tissue forms wherever destruction has occurred and it is concerned mainly with the absorption of dead bone. New bone formation occurs partly for the repair of the defect resulting from the death of bone and partly from the stimulative effect of the inflammation. The changes can best be considered under three heads: (1) New bone formation; (2) changes in the dead bone; (3) changes in the preexisting living bone.

1. New bone formation occurs about the dead bone and along the course of the living bone as far as the inflammatory process has spread. It occurs on both periosteal and endosteal surfaces. New bone forms along the peripheral surface of the dead bone, unless the periosteum has been destroyed by the infection or displaced following operation for drainage. The stripped off periosteum carries with it a considerable amount of osteogenetic tissue, which proliferates and forms a thick covering of fibroblasts, which at first show considerable evidence of inflammation. In from 5 to 10 days this layer begins to ossify, mainly by fibrous bone formation, although cartilage may appear in the process, as in the peripheral callus of a fracture. Ossification proceeds until a thick layer of spongy bone is formed, which is known as the involucrum. Periosteal new bone formation usually occurs along the living cortex for some distance beyond the dead bone. Its extent is dependent upon the spread of infection along the periosteum which does not result in necrosis of cortex. A thick layer of spongy bone usually forms, which is thickest where it meets the involucrum about the dead bone and tapers off as it recedes from the center of inflammation. If longitudinal incision for drainage is made early in the separated periosteum, it retracts and no involucrum forms in that region, leaving the dead bone exposed. If pus first ruptures into soft parts and is simply drained at operation without incision of the periosteum, involucrum completely surrounds the bone except

at points where periosteum has been perforated or detroyed. Sometimes large areas are destroyed, in which regions extensive defects will be left in the involucrum, and very rarely there is failure of involucrum formation in the entire circumference for a short distance. Usually there are a few various sized openings in the involucrum, known as cloacæ, through which the pus is discharged.

Endosteal new bone formation occurs in the medullary canal where the inflammatory process has spread, and may block the end of the shaft for some distance beyond the dead bone, as does the endosteal callus in a fracture. It also forms along the living portions of the shaft and from the cancellous bone of the interior, where the entire circumference of the bone has not been killed by the inflammatory process. Endosteal bone forms entirely by direct transformation of the fibrous into bony callus, cartilage never appearing in the process.

New bone formation continues for four to eight weeks, at the end of which time it is frequently voluminous. It then becomes denser and more or less laminated and is reduced to some extent in volume, similarly to the provisional callus found in the healing of a fracture. The extent of its reduction depends upon the amount of necrosis that has occurred and upon the duration of the inflammatory process. If the latter is gotten rid of, there is a gradual recession of the newly formed bone, but where the infection persists it may become eburnated and remain permanently.

2. Changes in the dead bone. Dead bone is absorbed only by the action of fixed tissue cells. Consequently it undergoes little change until the reparative processes are well established and a layer of granulation tissue forms about it. The small amount of connective tissue and blood-vessels contained in the bone are destroyed by the toxins and removed by the action of the serum and leukocytes. This destroys the pinkish color and gives it a white appearance, by which it may be distinguished from the surrounding living bone. Some time is required for this change in color to occur, and it is frequently difficult to distinguish between living and dead bone before the line of demarcation has formed.

The granulations attack the dead bone, separate it from the living, reduce it in volume and attempt to extrude it from the field through the discharging sinuses. Separation from the living bone is accomplished by the formation of grooves along the periosteal and endosteal surfaces, which are gradually deepened until they unite. It requires from a few days in the case of small bones to two or three months where thick cortex of a large bone is involved.

Reduction in volume of the dead bone occurs from lacunar absorption along its surfaces by the granulation tissue. The rate of surface destruction varies greatly and is dependent largely upon the relation of the surface to surrounding living bone and to channels of purulent discharge. Where living bone is in close contact with dead portions, granulations springing from and supported by it attack the dead bone, producing an irregular, worm-eaten surface, but where the dead bone lies at some distance from living bone and where the surrounding granulations are supported only by soft parts, the rate of absorption is much slower. Thus the periosteal surface of dead cortex becomes extensively eroded along those surfaces surrounded by involucrum, but remains smooth or little eroded for long periods where involucrum fails to form. This latter condition is seen in the regions of the windows in the involucrum, which are the result of local death of periosteum or incision with retraction of periosteum for the establishment of drainage. The condition of the endosteal surface varies with the degree of involvement of the shaft at any level. If less than half of its circumference dies, endosteal new bone will form along the living portion and support granulations in contact with the dead bone, destroying it from the endosteal surface, but if more than half the circumference is destroyed, there will frequently be little destruction of the deeper portion of the endosteal surface. When the entire circumference is destroyed, the endosteal surface of the shaft will remain unchanged for months or rarely for years, because the granulations cannot destroy it except at the ends. Where an involucrum surrounds a dead shaft, destruction from the periosteal side may finally lead to perforations of its cortex with ingrowth of granulations, after which endosteal erosion will occur. Where cortex is thick, as in the case of the middle of the shaft of the large bones, absorption may come to a standstill and be incomplete after years, because the space between involucrum and sequestrum becomes so wide that it is not bridged by healthy granulations. The rate of destruction is greater while the dead piece is still attached to or incarcerated by living bone. Once it is loosened and the sequestrum moves into a larger cavity or into the soft parts, the rate of destruction is greatly reduced. Destruction of necrotic cancellous bone and of thin cortex at the ends occurs earlier and more rapidly than that of dead cortex. This establishes drainage and gives rise to the first changes shown in roentgenograms. When not removed by operation, the sequestra may be extruded from the wound by action of granulation tissue and this process is apt to continue over long periods of time as small pieces become detached from larger sequestra. While large, compact sequestra in osteomyelitis of children may persist, they are usually so surrounded by involucrum that granulation tissue gradually destroys them or produces fragmentation and extrusion within six months to two years, if they are not removed by operation. Dead bone bordering on a sinus is slowly absorbed because it is bathed in pus from deeper portions which keeps the surrounding granulations unhealthy.

3. Changes in pre-existing living bone. The old living bone, bordering immediately on dead bone, undergoes rapid absorption by the granulation tissue. This process is known as caries and was observed by John Hunter to be of great importance in the process of sequestration. From spread of the infection to the surrounding living

bone for a variable distance, irregular caries may develop, leading to the formation of numerous small cavities, a condition known as rarefying osteitis. It may be responsible for the persistence of the infection and for attacks of recurring osteomyelitis. This stage may be followed by sclerosing osteitis if the infection becomes chronic.

Regional atrophy of the old bone results from disuse to which the part is subjected by the osteomyelitis. When the infection is limited and the loss of function is only partial and for a comparatively short time, the amount of atrophy may be slight. When the osteomyelitis is severe and loss of function is prolonged and complete, marked atrophy occurs. This reduces the density of the bone, which may be detected in from four to six weeks by means of the x-rays or at operation. With subsidence of infection and resumption of function, there is a gradual increase in density of the atrophied portion. Several months may be required for it to reach normal.

Transformation in the pre-existing bone occurs at the limits of the dead portion as a result of the realignment of the shaft with the involucrum. The lamellæ shift and become continuous with those of the involucrum in order to meet the new lines of force which pass by way of the involucrum. These changes are very marked where the course of the bone is greatly altered, either from bone destruction or by operative removal of living portions for the obliteration of cavities.

Cavity Formation.—Cavities arise in the medullary canal from new bone formation about infected areas and from rapid absorption of cancellous bone. Where dead bone exists in considerable amount and bony walls form about it, cavities result. At first they contain sequestra and these may persist indefinitely, but usually become reduced in size or after many months are broken up and discharged through sinuses. Cavities are lined by granulation tissue and contain pus while the sequestra are retained. When they are free of sequestra, fibrous tissue forms in greater abundance, partially or completely filling them, but it either fails to be transformed into bone or the transformation occurs very slowly. The reasons for this failure of the cavity to fill out with new bone are still imperfectly understood, but the main one is the sclerosed condition of the surrounding bony wall from which new bone formation takes place with great difficulty. This may lead to the persistence of fistulæ and predisposes to a recurrence of osteomyelitis, as bacteria are often indefinitely retained in the fibrous contents.

Sinuses may heal even though sequestra are still present, but the sequestra are usually small when this occurs and subsequent lighting up of the infection with reopening of the sinuses frequently results.

Diffuse Osteomyelitis with Little or No Pus Formation.—This type of infection is not infrequent and is insufficiently recognized. The point of onset and distribution of the infection is similar to that of the suppurative type, but it is usually less extensive. The striking feature of the infection is the absence or small amount of pus. The cancellous tissue of the end of the shaft, the bone marrow and periosteum are the seat of inflammation and considerable portions of cortex may undergo necrosis, but the exudate is small in amount and usually of a serous or sero-hemorrhagic nature. It may consist of a small amount of pus, which does not lead to perforation of the periosteum and abscess formation in the soft parts. Occasionally, the serous or sero-hemorrhagic exudate will be more extensive and break into the soft parts. New bone and granulation tissue formation occur as in the more extensive suppurating form and the dead bone when small is frequently entirely absorbed. This may lead to cavity formation and persistence of the infection with occasional exacerbations. It is not uncommon to see such cases run for years without abscess and fistula formation.

Where the infection is diffuse, necrosis of the cortex limited and new bone formation marked, the involved region frequently becomes enlarged and dense, a condition described by Garré as sclerosing osteomyelitis. Ollier described a non-suppurative process about the periphery of the shaft as albuminous periostitis.

Localized Osteomyelitis of the Shaft.—Localized infection of the shaft is common and shows considerable variation as to points of involvement and changes produced. The inflammatory area is usually small and circumscribed, leading to rapid breaking down of the centrally involved portion and the formation of a single cavity with a variable amount of sclerosis of the surrounding bony wall. This condition is usually called Brodie's abscess. The contents of the cavity consist early of pus and in some instances sand-like particles of necrotic bone. The pus may persist for indefinite periods, but usually with subsidence of the acute stage granulations grow in from the walls and partly fill the cavity. The pus may eventually disappear and the granulations organize so that the cavity is filled with fibrous tissue. With recurrence of the infection, which is not uncommon, the organized contents break down in part or wholly and pus formation again takes place.

Location of the Abscess.—The cancellous bone of the end of the shaft is most frequently involved, especially near the epiphyseal line. The entire process is usually subcortical, but the cavity may extend to the surface. New bone formation occurs in the surrounding cancellous portions, but is frequently small in amount. Periosteal bone formation is common where the abscess cavity approaches the surface. It usually consists of a layer of dense new bone, but may be laminated, as is so frequently the case in tuberculosis of this region.

When the shaft away from the ends is involved, the localization may be about the external or internal surface of the cortex. When internally located, abscess formation occurs in the medullary canal with new bone formation about its periphery, which partially or completely blocks the canal in the involved segment. There may be absorption or occasionally sequestration of adjacent cortex to a limited extent. By extension to the periphery, periosteal new bone formation may occur, leading to a spindle-shaped sclerosing osteitis. Peripheral or subperiosteal localization usually leads to a small area of bone destruction and marked surrounding new bone formation. The contents of the cavities located along the course of the shaft are variable, the endosteal ones frequently containing pus over long periods of time, while the small ones situated about the periphery are more apt to be filled with granulation tissue.

Bone abscess may persist for months or even years and pass through periods of exacerbation and still remain confined to the bone. In other cases there is extension or rupture into the surrounding soft parts, with the development of a localized indurated area of inflammation or abscess and fistula.

Chronic and Recurring Osteomyelitis.—Lighting up of inflammation at the seat of an old osteomyelitis usually occurs in the region of a cavity, sinus or sequestrum. Consequently the surrounding sclerosed bone is involved in the process and extensive spread of the infection does not usually occur. Limited suppuration, rarefying osteitis and new bone formation without the development of large sequestra are the usual result. Where the cortex is thickened and sclerosed and the medullary cavity has been previously involved, there is a marked tendency for the process to extend along the medullary canal and lead to the formation of one or more abscesses with internal sequestra, where pus may be retained and symptoms produced for long periods of time.

Typhoid Osteomyelitis.—Bone infection developing as a complication or sequel of typhoid fever usually produces the picture of localized osteomyelitis, similar, in location and changes produced, to localized osteomyelitis from pyogenic cocci. Occasionally the process has a tendency to spread, producing necrosis of a considerable area of shaft and subsequent sequestration. Perforation and abscess formation in the soft parts are common, and chronic fistulæ are frequently left.

Osteomyelitis Variolosa.—Aside from the occasional pyogenic osteomyelitis which may complicate smallpox as a result of pus organisms gaining entrance to the circulation through skin lesions, there are distinctive lesions of the bone which are apparently the result of the smallpox virus. Circumscribed areas of necrosis in the bone marrow occur frequently, but they are of no clinical importance and clear without leaving any after effects. Musgrave was the first to point out that the smallpox virus may affect the epiphyseal line in children and destroy the epiphyseal cartilage, resulting in fusion of the center of ossification in the epiphysis with the end of the shaft and arrested longitudinal growth. Several epiphyseal lines may be so affected, so that marked deformity of the extremities results. Brown reported one case in which there was arrested growth in eight locations. Figure 10 shows the result of obliteration of the epiphyseal line of the lower end of the radius in a 33 year old man, which resulted from smallpox at the age of three.

Symptoms.—There is great variation in the symptoms of osteomyelitis, according to the location and severity of the inflammatory change. Most cases have an acute onset with general and local symptoms of pyogenic infection, followed by a subacute or chronic course, dependent to some extent upon treatment.

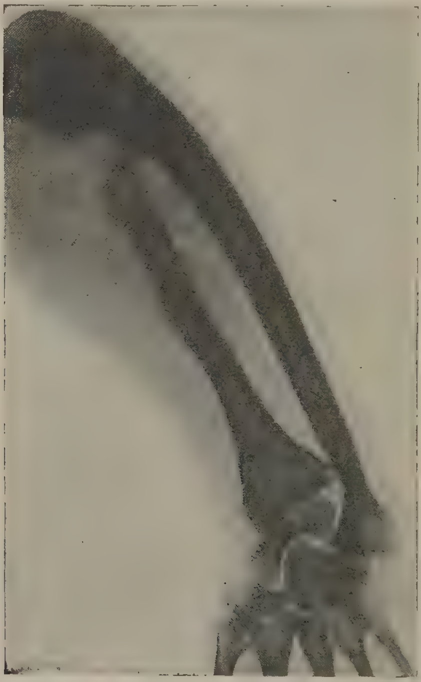

FIG. 10.—Arrested longitudinal growth of radius from smallpox osteomyelitis at age of three.

Diffuse suppurative osteomyelitis begins suddenly with local pain, which is severe from the onset and of a boring and throbbing character. It is usually located in the end of the shaft, but may extend along the entire course of the affected bone. The neighboring joints can usually be moved at first without pain, but loss of function soon develops, as movement of the involved extremity becomes painful. Marked general symptoms of infection usually develop soon after the onset of the pain and before the appearance of other local symptoms. A chill or chilliness is not uncommon, followed by rapid elevation of temperature. At first the involved bone may show no tenderness, but within 12 to 24 hours, depending upon its size, definite tenderness can usually be elicited upon deep pressure. Swelling soon appears and gradually increases. At first there is simply edema of the overlying soft parts, but this is soon accompanied by marked local heat and redness of the skin, with greater swelling in the regions denuded of periosteum, and extension of the infection to the soft parts. Where the process spreads along the course of the bone, extensive local changes accompany it in the overlying soft parts, and in case of involvement of the entire shaft enormous enlargement of the segment soon results. Fluctuation can usually be detected in from 3 to 10 days, but if the soft parts are thick it may be greatly delayed until the pus has perforated the deep fascia. Occasionally erythema of the overlying skin will be extremely marked toward the onset and suggest primary involvement of the soft parts. General symptoms usually increase during the period of local extension, the temperature remains high, ranging from 101 to 104 degrees, depending upon the size of the bone involved. The pulse is accelerated in proportion to the elevation of temperature. Toxic symptoms are present and delirium is not uncommon. In young children they may be marked from the onset and so obscure the local symptoms that pain is not complained of and the seat of the process is overlooked until swelling occurs. The face is frequently flushed, the tongue dry, the breath foul, and vomiting may occur. Leukocytosis is nearly always present and usually persistently high. Blood cultures are usually positive within two or three days of the onset and usually show staphylococci. Severe cases continue in this septic state and frequently end fatally within 5 to 10 days. The symptoms either remain stationary or progress until the height of the inflammation is reached in from 7 to 14 days. If drainage is established early, the septic condition is lessened, the duration of the severe acute symptoms is shortened, and frequently by the tenth to the fifteenth day the temperature shows only moderate afternoon elevation. If drainage is not established by operation and the overlying soft parts are thin, the pus may work its way to the surface and be spontaneously discharged. Rapid loss in weight and strength occurs during the septic period and if it continues much beyond a fortnight emaciation becomes marked. Where extensive infection persists as a result of very extensive bone involvement or from neglect of treatment, the patient passes into a low septic state which may last for weeks, during which period complications frequently develop. When adequate drainage has been established and the infection localized, the process passes into a subacute stage, during which period general symptoms are slight or absent. The local reparative processes are then accelerated, new bone forms rapidly and dead bone is rapidly absorbed. The purulent discharge usually continues but becomes less in amount and cicatrization occurs, leading to decrease in size of the wound and the establishment of sinuses. There is frequently increase in weight and strength after the process becomes localized, and when the osteomyelitis is not extensive, general health may be restored to normal within a few weeks.

Function is markedly interfered with during the height of the inflammation, and for long periods afterwards in case destructive changes are extensive, but usually begins to return as the process becomes localized. It remains impaired for an indefinite period, depending on the duration of the infection and the extent of damage to the soft parts.

Diffuse osteomyelitis in which little or no pus forms produces symptoms similar to but milder than those just described. If the involved area is not operated on early, the general symptoms are more apt gradually to subside than in the suppurative form, and a chronic condition follows.

Localized osteomyelitis or bone abscess produces a variety of symptoms. The onset is usually acute, with pain in the involved region, followed by mild general symptoms of infection. The pain continues moderately severe for a number of days and slight local swelling and tenderness develop, but abscess formation in the soft parts rarely occurs. The active period lasts from one to three weeks, after which general symptoms usually disappear and local signs are greatly reduced. The affected region usually remains slightly swollen, sensitive and painful. The pain is frequently more marked at night and may greatly disturb sleep. Recurrences are common at irregular intervals of two to six months, during which mild general symptoms recur and local symptoms are exaggerated. In the intervals there may be periods of complete freedom from symptoms. During a recurrence there may be extension to the periphery with abscess and fistula formation. It is extremely rare that a primary localized osteomyelitis subsequently gives rise to a diffuse process. Multiple foci may develop in different bones over the course of months or years.

Occasionally the symptoms may be chronic from the onset and show no fluctuation in character, in which case the cavity is usually small and at operation is found to be filled with granulations instead of pus. This form is often mistaken for syphilis.

Silent Foci.—Secondary foci of localized osteomyelitis not infrequently produce no local symptoms or signs at their onset and a fair sized area of bone may be absorbed and new bone may form about it before the lesion is detected. Such lesions eventually produce pain and tenderness, and an *x*-ray picture taken immediately after the first symptoms are complained of may show evidences of a lesion the result of an infection of weeks' or even months' standing. For this reason, every complaint of pain in the region of a bone in a patient with osteomyelitis calls for careful examination and a radiogram. In case of multiple foci of localized osteomyelitis, where some produce no local symptoms at their onset, roentgenograms of the entire body may reveal other foci that have as yet not produced symptoms. The author has discovered in this way as many as four foci in one patient.

Roentgenologic Findings.—The roentgenologic evidences of osteomyelitis are dependent upon the occurrence of bone destruction and new bone formation, both of which are well defined in the ordinary case; but in the exceptional cases one or the other change may predominate and give rise to difficulty in diagnosis.

In diffuse osteomyelitis bone destruction is usually the first change to be observed. Irregular reduction in density is seen in the cancellous bone and in the cortex of the end of the shaft, extending from ½ to 1 in. away from the epiphyseal line. It is first noticed in from 7 to 14 days, depending upon the size of the bone involved, the smaller bones showing the earlier change. Evidence of reduction in density in the compact bone does not appear for some time afterward, depending upon the thickness of the cortex involved and its relation to surrounding living bone. New bone formation from the involved periosteum produces a faint shadow along the cortical surface, which can be seen at the same time as or soon after the first evidences of reduction in density. Shadows from endosteal new bone may also appear at this time, but are usually seen later, due to the fact that they are feeble and are rendered less distinct by overlapping cortex. The peripheral new bone springing from living cortex casts a shadow that is less dense than and continuous with that of the bone from which it springs. On the other hand, the portion covering dead cortex casts a fainter shadow than the dead bone that it surrounds, but the two shadows are not continuous, being separated by a space, varying in width from 0.25 to 0.50 cm., which casts a very feeble shadow and represents the zones filled with granulations which surround the dead bone. Signs of irregular reduction in density appear in from three to six weeks along the line of junction of dead and living cortex. Frequently a notch is first seen in the cortical line, which subsequently deepens, and eventually a complete jagged zone of demarcation can be made out. The outline of the periosteal and endosteal surfaces of the dead bone may gradually become uneven, which is a result of absorptive action by the granulations. Surfaces uncovered by involucrum may retain their original sharp contour, as pointed out by Phemister. As the density of the surrounding new bone increases, that of the living old bone decreases. For a short distance away from the dead bone the density is sometimes irregularly reduced, due to rarefying osteitis. Beyond that, the reduction is more uniform and may even be longitudinally streaked from dilated Haversian canals. This reduction is due to bone atrophy, which arises from disuse. Frequently by the end of 6 to 10 weeks the reduction in density of the old living bone from rarefying osteitis and atrophy is greater than that of the dead bone produced by absorption of the granulation tissue. Consequently the dead bone at this time usually casts a heavier shadow than the old living bone, and, if not, its shadow is more uneven than that of the living bone, due to the irregular, worm-eaten structure of the latter.

The features by which the dead bone may usually be distinguished are its greater density, irregular contour, sharp outlines, pointed extremities and the presence of a surrounding zone of low density, demarcating it from the living bone. Older osteomyelitis shows greater density of the newly formed bone, and reduced density, indicating cavity formation, in the regions of bone destruction. Sequestra showing at a late date are usually much reduced in size and density. Sclerosing osteomyelitis may show regional enlargement and marked increase in density in both periosteal and endosteal regions without any signs of cavity formation.

Localized osteomyelitis or abscess usually results in rapid formation of one somewhat spherical shaped, central area of reduced density, which may be seen roentgenologically in 10 to 15 days, and becomes quite circumscribed in three to four weeks, as the absorptive changes come to a standstill. About it is a zone of increased density of varying degree. The density of the shadow is usually greatest about the abscess wall and gradually diminishes away from it. Occasionally, however, the periosteal shadow is laminated.

Complications and Sequelæ.—The complications of osteomyelitis consist largely of two groups, one arising from local and the other from hematogenous spread of the infection. *Arthritis* is a common complication, its frequency and severity varying greatly with the bone involved. Joint infection arises usually by extension of the process from the surface of the infected end of the shaft to the synovial membrane, less frequently from a secondarily involved epiphysis or from rupture of a soft parts abscess. Where the synovial lining is reflected along the ends of the bone, close to or beyond the epiphyseal line, as in the case of the ends of the femur, joint infection is very common. Osteomyelitis beginning in the neck of the femur practically always produces suppurative coxitis, as the neck lies completely within the joint and is surrounded by synovial membrane. Extensive destruction of the joint usually occurs and pathological dislocation is a common result. The knee, ankle and elbow are frequently involved, in which locations the exudate is usually serous or sero-purulent, but purulent arthritis resulting from direct extension from the end of the shaft or secondarily involved epiphysis or from rupture of a soft parts abscess may occur. Hematogenous involvement of remote joints is an occasional complication, arising oftenest during the height of the infection or as a complication of a later septic stage. The exudate is usually of a serous or sero-purulent nature, but may consist of pus and be of serious consequence.

Tenosynovitis by extension arises most frequently from osteomyelitis of the bones of the hands and feet, but is occasionally seen in the tendons of the forearm and leg from involvement of the bones of those parts. It is usually of a purulent nature and frequently results in loss of the tendon with marked stiffening and contracture. Bone felons affect especially the flexor tendons of the fingers.

Hematogenous tenosynovitis is an occasional complication.

Thrombophlebitis complicates most frequently the severe cases and may give rise to pyemia and death. In osteomyelitis of the femur and humerus it may lead to obstruction of the deep veins of the leg or arm with subsequent varicosities and enlargement of the extremity.

Pathological fracture occurs occasionally in the severe forms. It is most frequently due to extensive destruction of the end of the shaft, where rapid bone absorption occurs rather early, before periosteal new bone formation, which is least extensive in this region, has formed a supporting involucrum. The end of any long bone may be involved, but it is especially frequent in the neck of the femur, due to the fact that a poor osteogenetic covering exists in that location. This may be followed by complete absorption of the femoral head and marked deformity of the hip. Fracture of the shaft away from the ends is rare, and only occurs where there is extensive destruction of periosteum and death of the entire circumference of the shaft, in which case there is absence or incomplete formation of involucrum. It occurs some time after the sixth week, when sequestration is complete, and where there is not sufficient involucrum to support the limb. The involucrum occasionally bends at one end of the sequestrum, leading to persistent curvature of the bone. Fracture is not an infrequent complication of operation in the later stages, where considerable bone is removed.

Chest complications occur especially in the severe forms. Lung abscess may result especially from septic embolism, and empyema may arise from rupture of a peripherally situated abscess into the pleural cavity. Pneumonia and empyema may arise independently of gross embolic processes in the lungs. Pericarditis of the serous or suppurative form is a rare occurrence, as is also endocarditis. Secondary foci of osteomyelitis in other bones develop frequently. They are seen most frequently during the acute stage, but may arise at any time, and are usually milder than the primary focus.

Special complications arise from osteomyelitis of certain bones. When the skull is affected, extra-dural or brain abscess or meningitis may develop. Osteomyelitis of the spine may produce a variety of special complications, as shown by the collective studies of Volkmann. There may be rapid extension through or perforation of the dura with acute meningitis. Abscess or granuloma formation may occur in the spinal canal outside of the dura and press upon the cord, producing partial or complete paralysis below that level.

Bed-sores form in the severe and prolonged cases with marked emaciation, and when extensive may become a serious complication. Putrefactive infection and septicemia may follow extensive decubitus.

Disturbance in bone growth is not uncommon. If the cartilaginous epiphyseal line is completely destroyed, arrested longitudinal growth will result in marked shortening of the bone. Occasionally it is destroyed in part and arrestment of growth occurs only on the affected side which produces curvature of the bone. Lengthening of the affected bone may also occur, and is most frequent where the osteomyelitis is extensive and the epiphyseal lines uninvolved; also where the infectious process becomes chronic. It is the result of the stimulative effect of the infection upon bone growth, and may continue for years in case the disease persists. Not only the affected bone, but the neighboring bones of the involved extremity may be stimulated to excessive longitudinal growth. This is especially the case where the affected bone is in the proximal part of the extremity, as overgrowth of tibia and fibula in osteomyelitis of the femur. From ½ to 1 in. of lengthening may be observed in either affected or neighboring bones.

Diagnosis.—Errors in diagnosis occur much more frequently in osteomyelitis than should be the case. This is due to imperfect analysis of the symptoms, inaccuracy in physical examination and wrong interpretation of roentgenologic findings. Errors are especially common at the onset, during which period roentgenograms are negative. Because of the general symptoms it may be mistaken for typhoid fever, meningitis and other severe infections. Because of the local symptoms it may be mistaken for rheumatic fever, acute arthritis, cellulitis of the soft parts, deep lymphangitis, thrombophlebitis or erysipelas. The late cases may be confused with tuberculosis, syphilis, osteitis deformans cyst, or even tumor of the bones.

The severe forms with marked general symptoms should be distinguished from typhoid by the leukocyte count, blood culture and Widal test. Physical examination soon reveals localized signs of inflammation, even though no local symptoms are complained of. The eventual development of local findings make it possible to distinguish osteomyelitis from other severe infections.

Rheumatism is the condition for which it is most frequently mistaken. The onset of osteomyelitis is usually more abrupt and the local pain more severe and referred to the end of the shaft rather than to the joint. In the great majority of cases one region is involved in osteomyelitis, while in rheumatism several joints usually become affected. The temperature is higher in osteomyelitis, and leukocytosis is high, while in rheumatism it is rarely above 15,000. The swelling and tenderness in osteomyelitis extend away from the joint, while in rheumatism they center about the joint. Even where arthritis develops by direct extension, the joint symptoms are of minor importance in comparison with those associated with the involved bone. The development of pus or enlargement of the bone is conclusive evidence against rheumatism.

Cellulitis or abscess formation arising in the over-lying soft parts may be confused with abscess developing from infected underlying bones. The local wound or infection of the skin, through which microörganisms have gained entrance, is usually demonstrable in such cases. Because of the early absence of roentgenologic changes in both conditions, an exploratory incision which demonstrates the absence of a connection with the bone may be necessary in order to make out the distinction.

Tuberculosis, syphilis, cysts and tumors are confused oftenest with localized osteomyelitis and with the diffuse cases which do not lead to abscess and fistula formation. The history of an acute onset with general and local symptoms, and of acute exacerbations in the subsequent course of the disease is strong evidence in favor of osteomyelitis. In osteomyelitis there is usually about the same degree of both bone destruction and new bone formation, in tuberculosis bone destruction is apt to be greater than new bone formation and the newly formed periosteal bone is not infrequently laminated in character; in syphilis new bone formation is usually more extensive than bone destruction, and the areas of bone destruction are very uneven in size and irregularly distributed. The presence of dead bone in considerable amount is in favor of osteomyelitis, although tuberculosis may produce sequestra of fair size. There are no distinctive findings for any one of the conditions, and histological and bacteriological examinations are occasionally necessary for the final diagnosis.

Cysts and myeloid tumors may simulate osteomyelitis, but the predominence of destructive changes in these lesions and the absence of local and systemic evidences of infection are usually sufficient for diagnosis.

Prognosis.—The prognosis as to life varies greatly with the form and treatment. Severe cases that remain unrecognized frequently die in a short time with the picture of marked sepsis. Cases that survive the acute period and are adequately drained rarely succumb unless complications develop. The prognosis as to duration is extremely variable and dependent upon the severity of the disease and upon the treatment received. In the most favorable cases of the diffuse form, several months usually elapse before healing is complete. In the localized forms, where sequestra are absent, and when properly treated, healing may be complete in from 6 to 10 weeks. Persistence of the infection with recurrence of symptoms, is dependent mainly upon the presence of cavities and of chronically infected bone. It is never possible to state with certainty that osteomyelitis is cured, as recurrence is not infrequent following the most appropriate operative treatment and after the lapse of many years, where physical and roentgenological examinations show an almost complete absence of bony change.

Treatment.—All forms of osteomyelitis, when seen early in the course of the disease, should be treated by immediate operation for establishment of thorough drainage. This is especially true for the diffuse form, where delay results in spread of the infection with greater local destruction, persistence of general symptoms and increased danger of the development of generalized infection or serious complications.

Early Operations.—When possible the operation should be done with a constrictor applied, in order to lessen the amount of hemorrhage and determine more accurately the extent of involvement. The incision should be placed so as to establish free drainage, but at the same time the soft parts should be respected as much as possible. The classic approach for the various bones of the body, which produces the least amount of injury to muscles, nerves and vessels, should be adhered to where extensive incisions are necessary in severe cases. This is well given for each of the bones in the work of J. E. Thompson. In early cases the soft parts are incised to the periosteum. Pus may be encountered beneath or between the muscles. If rupture has not taken place, the periosteum will be distended, and, when incised, pus will escape from the space between it and the bone. A variable amount of separation will have taken place. In mild cases it will be slight and limited to a part of the circumference of the end of the shaft. In other cases it will be more extensive, involving the entire circumference of a section. In still other cases the entire length of the bone will be involved in a part or all of its circumference. The periosteum should be incised for the length of the separation. In diffuse osteomyelitis drainage of the interior of the bone should be established by the excision with a chisel of a strip of cortex sufficiently long to expose the cancellous bone and the first portion of the medullary canal of the end of the shaft. This is sufficient for the escape of pus from this end. If the inflammation has spread the entire length of the shaft and involved the other end, the procedure should be repeated there. It is unnecessary to remove cortex along the middle portions of a necrotic shaft to get drainage. Because the pus frequently lifts up the periosteum for variable distances without the occurrence of death of the underlying bone, it is unwise to excise a strip of cortex the entire length of the periosteal separation, as living cortex may be damaged thereby. The medullary contents should not be curetted, as in case they are not all dead and part of the circumference of the shaft is alive, bone forming elements may be unnecessarily removed. After hemostasis of the soft parts, the wound should be packed with gauze and left open. The wound should be dressed in from 24 to 48 hours, and frequently for the first time under nitrous oxide anesthesia, as the packing adheres and its removal is very painful. There is much difference of opinion as to the best method of subsequent care of the inflammatory area. The commonest form of treatment consists of loosely packing the wound with gauze down to the bone and frequent changing of dressings, depending upon the amount of discharge. Moist, hot normal salt solution dressings, changed three or four times a day, are comfortable for the patient and favor free drainage. Irrigation once or twice daily with normal salt solution or a mild antiseptic may occasionally be helpful, if there are poorly drained pockets and much pus, but its importance in the ordinary case is frequently over-estimated. Frequent changes of dressings may produce too much pain, in which event dry dressings, changed once or twice daily, may be indicated. A drainage tube introduced to the bone may be indicated, if the overlying soft parts are thick and have a tendency to close. Antiseptic treatment of osteomyelitis has been revived as a result of its use in war wounds. The Carrel-Dakin treatment is the one most frequently employed, and can be instituted as soon as drainage has been established. It is very questionable if the results from its use are superior to those obtained from the use of frequently changed moist or dry dressings.

If the process is extensive and located in the lower part of the femur or in the tibia, the patient may be more comfortable if the limb is supported in a Thomas splint, as the dressings can then be changed and the wound irrigated, if desired, without moving the limb. After drainage is established, the general and local symptoms as a rule gradually subside and the temperature approaches normal within a few days. If fever persists and local symptoms continue, the involved region should be watched carefully for evidence of extension of the infection in the bone and soft parts. In case this occurs, operation should again be resorted to and adequate drainage established.

The adjacent joints should be watched for evidences of arthritis, and treated appropriately in case it develops. In purulent arthritis weight extension should be applied and the joint opened and allowed to drain, but where the exudate is serous or sero-purulent, frequent aspiration, or aspiration followed by irrigation, may obviate the necessity of arthrotomy. Mild arthritis should be left alone.

Early operation for localized osteomyelitis should consist in extensive sloping removal of bone in the region of the infection, so that a deep cavity is not left. Healing follows in the majority of cases, and a secondary or late operation is usually not indicated, when the first intervention is properly performed.

Intermediate Operations.—These include the operative procedures that may be indicated during the subacute stage, before sequestration is complete or before a strong involucrum has formed. Operative removal of the dead bone during this period may be performed for two reasons. Where necrosis is very extensive, as in death of the entire shaft of a long bone, severe infection persists despite free incision. In this case, subperiosteal resection of the shaft may be done in order to get rid of the infected focus. It may also be performed in extensive cases, where the general symptoms have subsided, with the idea of rapidly getting rid of the necrotic bone and lessening the danger of cavity formation, thereby shortening the convalescence and lessening the chances of recurrence. The work of Nichols placed the procedure on a sound basis. It should not be performed on a bone that is the sole support of a segment of an extremity, such as the humerus or femur, except as a life-saving measure. The reason is that the involucrum which forms before sequestration is complete is too weak to support the extremity, and fracture with curvature, shortening and occasionally bony defect may result. In the forearm or leg, where a second or splint bone is present, subperiosteal resection of the involved region, carried beyond the limits of the dead bone, is in many cases an operation of very great advantage. Where dead bone is extensive, irregularly distributed and inaccessibly situated, as on the posterior side of the tibia, it more completely assures the removal of all dead bone and lessens the likelihood of cavity formation and the persistence of fistula. The time at which it should be performed varies with the bone involved and the rapidity of involucrum formation. The most appropriate period is between the fifth and eighth weeks, after a thick involucrum has formed, but while it is still composed of spongy bone which is pliable and can be reflected from the dead bone and detached with a sharp periosteotome from the surviving portions of the shaft. It can even be done much earlier with success. The soft tube which remains may at times be collapsed into a thick band by means of interrupted catgut sutures. At the ends of the shaft the separation is made along the epiphyseal line, but the cartilage is preserved, and after the involucrum has been converted into a new shaft, longitudinal growth continues. Cavity and fistula formation are not uncommon in the metaphyseal regions, because the involucrum cannot collapse at its epiphyseal attachment and completely fill in the space left by the removal of the dead bone. Amputation may occasionally be indicated as a life-saving measure in very extensive infections, especially where suppurative epiphysitis and arthritis are present.

Late Operations.—These consist in operations that are performed after sequestration is complete and after sufficient new bone has formed to support the extremity in case its support is perilously weakened by the loss of old bone. The objects of these operations are (1) the removal of dead bone, (2) the effacement of cavities, tunnels and sinuses, and (3) the removal of chronically infected living bone. The first two of these procedures are the ones of importance, and if they are successfully carried out, infection in the remaining living bone usually subsides.

Mild cases of osteomyelitis may not require late operation, as such bone as dies is absorbed or discharged, and cavities may either not form or fill out with new bone. However, recurrence is not rare in cases that have healed spontaneously after the establishment of drainage.

Sequestra should always be removed, unless they are extremely small and not associated with cavity formation. The time for removal of dead bone depends to some extent upon the surrounding living bone. If it is sufficiently strong to support the part, as in case of death of only a portion of the circumference of the shaft, removal should be performed as soon as sequestration is complete. This varies from three or four weeks, in the case of small bones, to three months, in the case of thick cortex of large bones. If a portion of shaft is dead in part or all of its circumference, sequestration may be complete before the involucrum is sufficiently strong to support the part alone. In this case it is necessary to leave the dead bone until the portion of involucrum to be left after operation is sufficiently ossified to support the part.

Procedures for obliteration of cavities, sinuses and tunnels should usually be carried out at the time that the dead bone is removed. They are numerous. The most important are (1) sloping removal of the obstructing bony walls, permitting the entrance of soft parts into the cavity, (2) filling the cavity with pedunculated flaps of muscle or transplanted fat, and (3) filling the cavity with antiseptic foreign substances, as Mosetig Moorhof bone wax.

The regions of dead bone and cavities should be widely exposed and the soft parts reflected, but care should be exercised not to separate them needlessly from living bone that is to be left, as fresh infection of the denuded portions of bone may occur. Involucrum should be removed the entire length of the involved segment in approximately one-half of its circumference. The dead bone is then extracted and the granulations are curetted out in order to assure the complete removal of all minute sequestra and the exposure of cavities and fistulæ. The most satisfactory way of obliterating cavities and fistulæ is by the sloping removal of sufficient bony wall to allow soft parts to fall in and fill out the remaining space. In extensive osteomyelitis of humerus or femur, three to five months should elapse before this operation is performed. The earlier the dead bone is removed and the softer the involucrum, the less the tendency to cavity formation. Care must be exercised not to weaken too greatly the shaft, as fracture may follow. This is often the case when a tunnel exists and one of its walls is removed. Sometimes it is advisable to remove the dead bone and leave nearly all or all of the living bone to hypertrophy, and operate if necessary at a subsequent date for obliteration of cavity or tunnel. Pedunculated flaps of muscle are especially indicated in large cavities near the ends of the bone, or occasionally in tunnels where the removal of one wall would greatly weaken the bone. Free transplants of fat are more likely to slough out. Mosetig-Moorhof bone wax if practically always discharged if inserted in cavities at the time of removal of sequestra, and fistulæ are much commoner after its use than following removal of walls for the effacement of cavities.

Old cases of osteomyelitis, in which cavities are present surrounded by sclerotic bone, are best treated by sloping removal of bony walls, but when filled with bone wax after a thorough cleansing, they may heal by primary intention and the mixture be gradually replaced by fibrous tissue or bone.

Cavities at the ends of the shaft are especially difficult to fill with soft parts, because of the large size of the bone and their nearness to the joints, which precludes complete effacement of their walls. They may be successfully filled by pedunculated flaps of the surrounding skin and subcutaneous fat. This procedure is particularly suited to cavities of the ends of the tibia. Skin grafts may be used successfully in such cases.

Chronically infected bone, which causes the persistence of fistulæ or the recurrence of acute infection, may be extensively removed, even though cavities of appreciable size and sequestra are absent. It is difficult to determine the limits of infected bone in such cases, and if operation is resorted to, excision of bone should be extensive.

Localized osteomyelitis or Brodie's abscess is best treated by extensive sloping removal of the abscess walls and thorough cleaning out of the cavity. Occasionally the focus will be so located that the region can be excised in toto. If the infection has stood for several months and the inflammatory process is quiescent, the wound can usually be closed without drainage and primary healing obtained.

Primary Periostitis.—Primary involvement of the periosteum is a rare occurrence as compared with secondary involvement by extension from the shaft. In case the inflammatory process is located mainly about the periphery of the bone, affecting both periosteum and cortex, it is difficult to know which structure was originally affected. The etiology does not differ from that of osteomyelitis of the shaft, except that the bacteria are frequently more attenuated and localization at this point is commoner in septic processes, as typhoid, small-pox and as a secondary focus in primary osteomyelitis of other bones. The periosteum toward the end of the shaft is most frequently the primary seat. The inflammation results in a subperiosteal exudate, which may be serous or purulent in nature, and separates periosteum from shaft for some distance about the point of onset, but rarely for more than 3 or 4 in. along the course of the shaft, and then only in a part of its circumference. The cortex becomes involved in varying degrees. Usually superficial necrosis with subsequent absorption of bone and occasionally sequestration occurs. The periosteum, which is dissected loose by the exudate, may be perforated in the suppurative form, leading to abscess formation in the soft parts. The exudate is usually retained when it is serous in nature and becomes mucous or stringy in character as absorption takes place. This form was first designated by Ollier as albuminous periostitis and later reported by Poncet. The periosteum forms a layer of new bone, which is similar to involucrum in case of osteomyelitis of the shaft. If there is no superficial necrosis of shaft, a cavity usually does not form beneath the layer of new bone; but if necrosis takes place, a flattened cavity remains between involucrum and shaft, filled with granulations which erode the dead bone. Sclerosis of the walls of the cavity may occur and the inflammation pass into a chronic stage.

The symptoms are usually mild, and frequently with an insidious onset. There is pain along the course of the involved bone, with a variable amount of swelling and tenderness, and the general symptoms of infection are slight, resembling those of bone abscess. Where pus forms, an abscess may appear in the overlying soft parts in the course of a few days. Definite, spindle-shaped bone enlargement may be made out in from two to four weeks, due to new bone formation from the involved periosteum. The process usually subsides gradually and entirely clears up, although a chronic discharge with recurrence may develop in case of cortical necrosis and superficial cavity formation.

Operation should be performed if the condition is seen early and the symptoms are at all severe. The separated periosteum should be incised and drainage established. If seen late and x-ray pictures show periosteal new-bone formation, superficial necrosis of the shaft and cavities, an operation should be performed with removal of the superficial layer of new bone, sequestrectomy and cleaning out of granulations. Care should be exercised that medullary involvement from extension of the process through the cortex is not overlooked.

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DISORDERS OF BONE FORMATION

Normal growth of bone occurs through the processes of enchondral and fibrous bone formation. Enchondral ossification or bone formation through cartilage is the process by which the epiphyses ossify and longitudinal growth of the long bones occurs. In it the cartilage bordering on the zone of ossification undergoes metamorphosis, forming a columnar zone of calcification. Capillaries, osteoclasts and osteoblasts grow into the zone of calcification, which by osteoclastic action is absorbed and by osteoblastic action is replaced by new bone. Enchondral ossification proceeds according to inherent tendencies that are little influenced by external factors, as stress and strain and the action of the surrounding soft parts. This is well shown by the fact that paralysis of a limb during the growing period, removing the influences of muscular action and weight bearing from the bones, retards but slightly their longitudinal growth. Fibrous or intramembranous bone formation is the process by which ossification of the bones of the vault of the skull and transverse growth of the shafts of the long bones occurs. In this process osteoblasts are transformed directly into bone without the formation of a cartilaginous medium. Fibrous bone formation is greatly influenced by the action of external factors. Thus in the case of a paralyzed growing extremity, marked arrest of transverse growth occurs, resulting in the formation of narrow shafts with thin cortices.

Disorders of bone formation arise as a result of congenital disturbances that are very little understood. In some conditions there is a difference in the degree of involvement of the processes of enchondral and fibrous bone formation. There is a definite relation between bone growth and function of the endocrine glands, but no one gland can be considered as the growth organ for the skeletal system. Physiological bone growth is dependent rather upon the normal functioning of all of the glands of internal secretion. The disorders may be classified according to whether the greater amount of disturbance is in the osteoblast or in the cartilage through which ossification occurs. Primary disturbance of the osteoblast gives rise to the condition of *osteogenesis imperfecta,* while primary disturbance of the cartilage leads to *chondrodystrophia fetalis, multiple cartilaginous exostoses* and *multiple enchondromas.*

OSTEOGENESIS IMPERFECTA

Osteogenesis imperfecta is a skeletal disturbance resulting from a deficiency in the osteoblasts, which leads to the formation of defective bones and to the occurrence of multiple fractures and deformities. It is also known as osteopsathyrosis and fragilitas ossium. Attempts have been made to separate the condition of bony fragility into a number of diseases according to the age of onset of the fractures, but it is best considered under one head.

Etiology.—The underlying cause is unknown. Heredity plays little part in the congenital form, but in the infantile and late forms it is of considerable importance. Conrad and Davenport report that a parent who is, or was in early life, osteopsathyrotic will have one-half his children affected if he marries a normal individual, but if his consort also be osteopsathyrotic three-quarters of the children will be affected. The author has studied a late case in which the disease extended through three generations, affecting the mother and four of her six children; an aunt and a nephew were also affected. Osteomas of the skull occurred in three of the members who were subject to fractures and in two who were not thus subject. Eddowes first noted the occurrence of blue sclerotics with osteopsathyrosis. Bronson has studied two families in which the conditions were associated. In a family of 55 persons in four generations, there were 21 with gray-blue sclerotics, and 20 of these had multiple fractures. In a second family of eight persons in three generations, seven had blue sclerotics and four of these had multiple fractures. It has been considered a primary skeletal disorder, but Kienböck, Hartmann, Bamberg and Huldschinsky have more recently regarded it as the result of defective internal secretion. Fragility of the bones results from complete thyroidectomy, and after removal of parathyroids, as shown in the experiments of Erdheim. Bone atrophy and curvatures developed in dogs thymectomized by Klose. However, no changes in the glands of internal secretion have been demonstrated at autopsy on cases of osteogensis imperfecta. Hart made pathological studies of a case of marked obesity and late osteopsathyrosis in a boy dying at the age of 12. Typical pathologic changes of the condition were noted in the bones, but there was no alteration in any of the ductless glands. Sumita has reported three cases in which the thyroid was carefully examined and found to be normal. The condition has not been found associated with other lesions at birth, but occasionally rachitic changes have developed during infancy. Malformations such as club foot, hydrocephalus and spina bifida are rarely present.

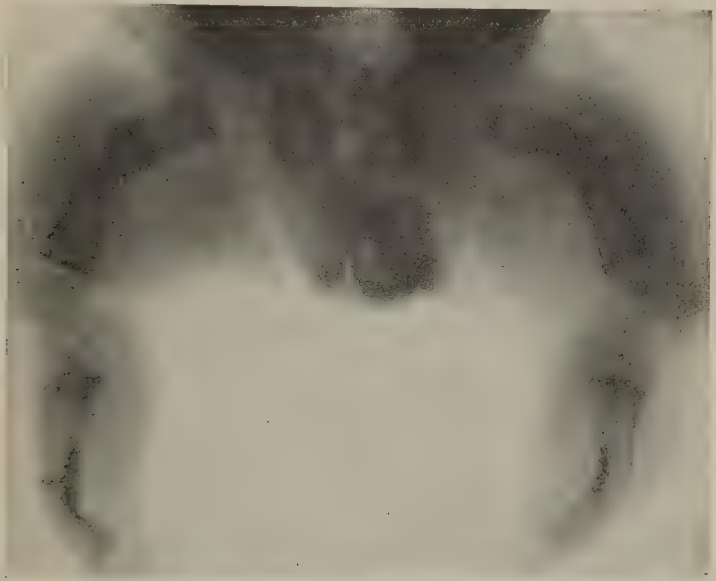

Fig. 11.—Osteogenesis imperfecta at birth, showing fractures in various stages of repair.

Clinical Manifestations.—These are extremely variable, depending upon the degree of involvement. Three fairly distinct forms, the congenital, infantile and late, have been described, according to the time of onset of the symptoms.

Osteogenesis imperfecta congenita is the commonest and severest form of the disease. It develops early in intra-uterine life, and most of the affected children are either stillborn or die shortly after birth. The appearance at birth is characteristic. The head is of normal size, seldom is it abnormally large. The cranium is frequently soft and crepitates in spots as a result of imperfect ossification of the bones of the vault. Ossification may be so slight that the head feels like a tense bag and changes its form with changes in position. Hair is usually abundant, the face is well formed, and its features are delicate. There is no thickening of the skin of the eyelids, lips and cheeks as in cretinism and chondrodystrophy, and the nose is not saddle-shaped. The neck is short and somewhat swollen. The trunk is usually normal in shape, but the abdomen is somewhat dis-

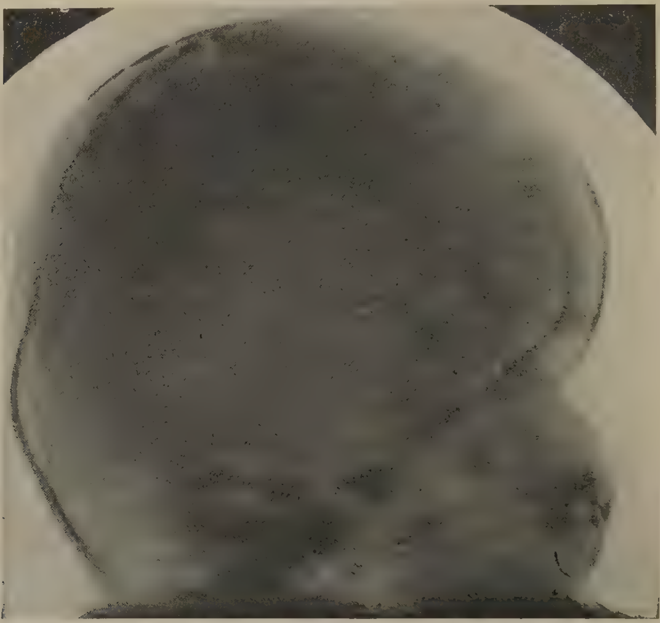

FIG. 12.—Skull of case in Fig. 11, showing mosaic along sagittal suture.

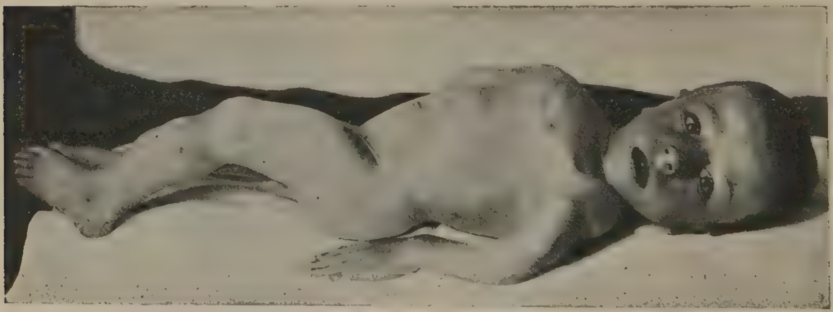

FIG. 13.—Osteogenesis imperfecta congenita at age of ten; weight 30 lb.

tended. The subcutaneous fat may be slightly increased, and deep folds traverse the thorax, connecting the axillary regions. Numerous nodules may be palpated along the course of the ribs and occasionally slight thoracic deformity is present. The spine and pelvis usually show no deformity and approximate the normal length and size. The extremities are often very short compared with the trunk, which makes the body length considerably below the normal. The arms and legs are stubby, but not actually misshapen. Curvatures and angulations may be irregularly distributed in the segments of the limbs. These are the result of fractures that have occurred at various times during intra-uterine life. The older ones may be firmly healed, while the more recent ones show both crepitation and false points of motion. The hands and feet are small and slendor as compared with the stubby condition seen in chondrodystrophy, and fractures are occasionally present.

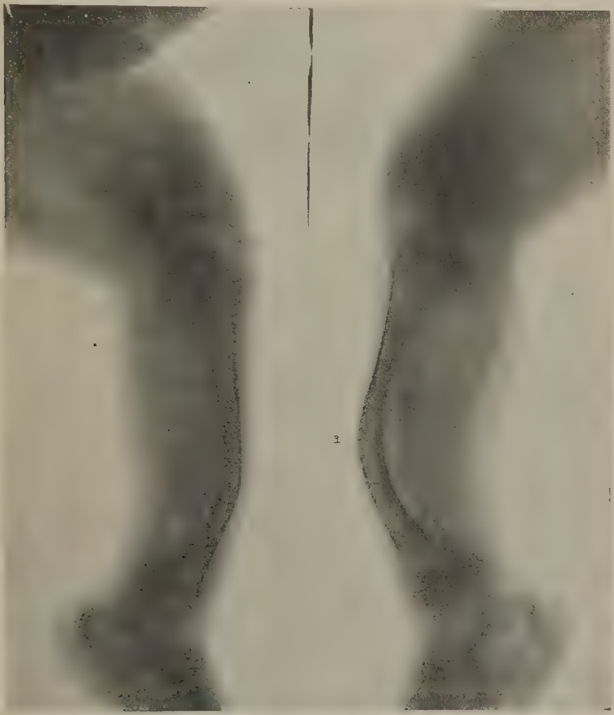

Fig. 14.—Roentgenogram of case in Fig. 13 at age of eight. (*a*) Transverse line marking seat of old fracture. Epiphyseal lines of femurs obliterated.

Surviving cases are comparatively rare. They become invalid dwarfs with deformed chests and frail extremities which are greatly deformed from the occurrence of repeated fractures. There is no great impairment of mentality, but lack of mental development because of the physical handicaps which they suffer. Figure 13 is of a boy 10 years old, weighing 30 lb., in whom fractures were present at birth. He could not walk and was unable to sit until the sixth year.

FIG. 15.—Roentgenogram of case in Fig. 13 at age of eight.

Handling and other slight violences had produced numerous fractures, only one of which, in the left humerus, had failed to heal promptly. He was fairly intelligent.

Osteogenesis Imperfecta Infantilis or **Infantile Osteopsathyrosis.**—In this form the children appear normal at birth and usually during the first year of life. After learning to stand or walk they develop fractures in the extremities, which are either spontaneous or the result of slight injury, indicating the fragility of the bones. Deformities develop according to the care with which the fractures are treated. Slight fractures or fissures frequently occur without being recognized, as the pain is often very slight and out of all proportion to the severity of the lesion. This results in neglect of the condition, which leads to the development of deformities. Marked bending and bowing are the result of fissure fractures of the long bones, and curvature of the shaft about the infraction is frequently so extreme as to suggest an associated rachitic or osteomalacic change. The lower extremities are much more extensively involved than the upper. Figures 17–19 are of a girl whose femurs had been repeatedly fractured; the bones of the upper extremities, however, although their cortices were abnormally thin, had remained free from fractures. The bones of the legs showed thin cortices and the upper end of the left tibia was the seat of an incomplete fracture which had produced no symptoms and was discovered only by the roentgenologic examination.

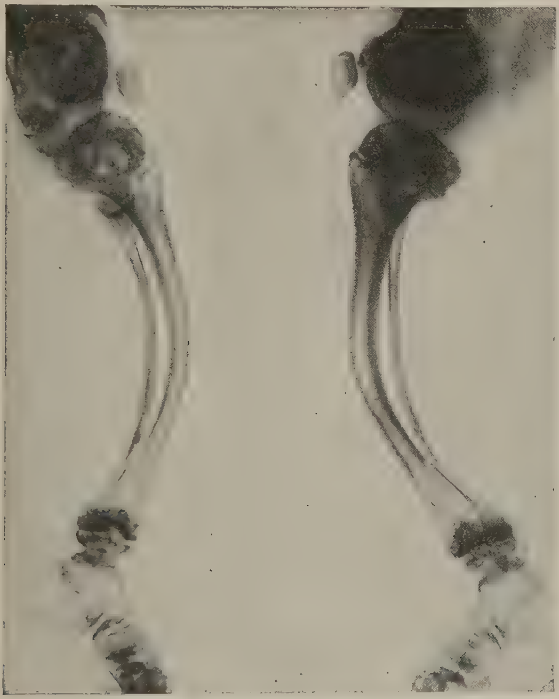

FIG. 16.—Same as Fig. 14 after four years of irregular treatment with phosphorus.

The femur usually shows the greatest tendency to involvement, and the humerus the least. Terry reported a case in which the upper extremities had been repeatedly fractured, the fingers as a result of handshaking. This case shows how fractures of the end of the shaft or of the epiphysis may lead to ankylosis of the neighboring joint. Separation of the epiphyseal line may cause arrested longitudinal growth. Most of the fractures heal, but pseudarthrosis occasionally results. Refracture of incompletely ossified calluses is not infrequent.

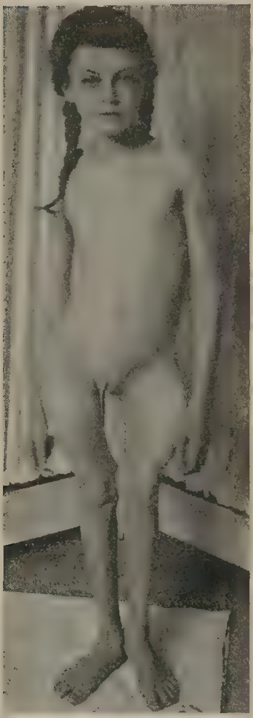

Fig. 17.—Osteogenesis imperfecta infantilis, eleven years old. Fractures limited to lower limbs, began at age of two. Patient of Dean Lewis.

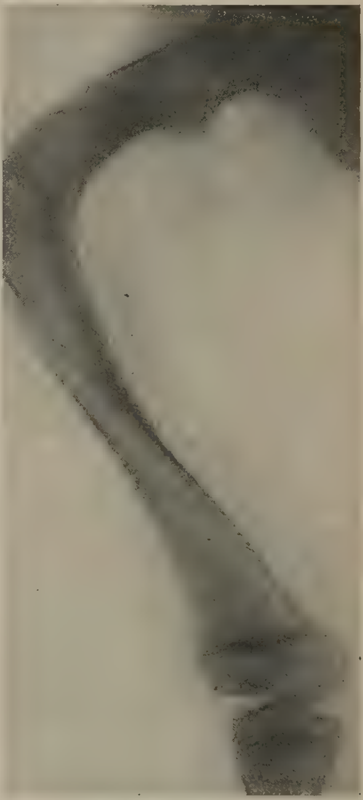

Fig. 18.—Roentgenogram of left femur of case in Fig. 17.

Such children are apt to be undersized and poorly nourished as a result of inactivity produced by the fractures and deformities. But dwarfism is extremely rare. They are susceptible to intercurrent diseases and frequently succumb before adult life is reached.

Osteogenesis imperfecta tarda makes its first appearance during childhood or adolescence. There is frequently a history of heredity and numerous families have been reported in which there is a tendency for the occurrence of fractures, which extends late into life. There is usually nothing abnormal during the first few years, after which fractures develop as a result of slight or moderate violence. They usually heal without any special tendency to pseudarthrosis and are distinguished with difficulty from fractures occurring in normal persons. Not infrequently the same bone will be fractured on different occasions, in which event deformity usually results. These individuals are usually of normal stature and their musculature may be well developed. Physical and roentgenographic examination shows frail bones of normal length which may be the seat of old or recent fractures, but without the curvatures seen in the earlier forms of the disease. Occasionally the pre-disposition to fractures disappears during the period of adult life while in other cases it does not set in until after adult life is reached. The author saw an extreme case in a man of 55 which began at the age of 40.

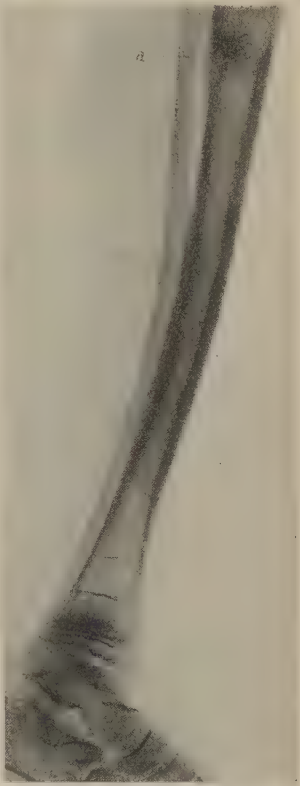

Fig. 19.—Imfraction (*a*) of right tibia in case in Fig. 17. Transverse lines in lower end of tibia due to previous phosphorus administrations.

Pathology.—The pathology is essentially the same in all three forms, but the changes are more extreme in the congenital and infantile forms. The disease is a deficiency of formation on the part of the osteoblasts, affecting especially the transverse growth of the shafts and ossification of those cranial bones preformed in membrane. Longitudinal growth of the shafts and ossification of the epiphyses suffer much in the congenital form, but are less markedly disturbed in the later forms.

In **osteogenesis imperfecta congenita** the cranial membrane shows irregular points of ossification at which thin bone plates develop. In some regions they may be fused, but in others they are arranged in mosaic, while in still others they may be absent, especially about the sutures and fontanelles (Fig. 12). This may give to the head the feeling of a hydrocephalus. The base of the skull is more completely formed, the separate bones being ossified, but they are very thin and easily broken or cut with a knife. Special attention has been given to the os tribasilare. It is not abnormally short and small centers of ossification are present. Its cartilaginous epiphyseal lines are of considerable width in fetuses examined at birth. The facial bones are as a rule almost entirely cartilaginous. The ribs are slender and narrow with a very thin cortex and relatively large marrow cavity. They are slightly enlarged at the costo-chondral junctions. The nodules scattered along the course of the ribs represent fractures in various stages of healing. Thickenings in the clavicle similar to those of the ribs may be present, and the scapula not infrequently shows signs of fracture. The bodies of the vertebræ are approximately of normal size and their centers of ossification are large but consist of brittle bone. Recklinghausen has seen reduction in the height of the bodies of the thoracic vertebræ leading to spinal curvature. Fractures of the spine and pelvis have not been recorded in osteogenesis imperfecta, but a slightly contracted pelvis has been reported by Hecker and Sandifort.

The extremity bones are usually soft and brittle and are frequently fractured upon endeavor to remove them at autopsy. In general they are soft, stubby and cylindrical shaped, and many are curved or angulated from previous fractures. There are numerous thickenings from callus formation at the seats of fractures. The epiphyses are either of normal size or somewhat smaller than normal. Upon section the bones show marked thinning and brittleness of the cortex, which even in the large bones may be as thin as an egg shell. There is no regularity about the thickness of the cortex, which in a normal bone is thin toward the epiphyses and gradually increases in thickness toward its middle portion. In osteogenesis imperfecta the cortex is irregular and may be as thin toward the middle of the shaft as in the metaphyseal regions. The medullary canal is filled with marrow, which in some areas is normal, but in others appears gelatinous. Considerable fat may be present. The periosteum is of irregular thickness, depending upon the presence of fractures, and subperiosteal hemorrhage may be present if a fracture is recent. The spongy bone of the ends of the shaft is scanty, the line of junction of shaft and epiphysis is straight and the epiphyseal cartilage is normal in appearance. Centers of ossification in the epiphysis usually appear at the proper age (Fig. 11). The fractures show moderate periosteal and endosteal callus formation, according to the extent of fracture and displacement. The callus may be fibrous, cartilaginous or bony, according to the age of the fracture, and non-union of old fractures is comparatively rare.

The later pathology of the surviving cases of the congenital form is similar to that of the infantile form, except that the involvement is more severe. There is general rarefaction of the bones, but less disturbance of longitudinal than of transverse growth. Consequently the shafts of the long bones are relatively long and slender and the cortices thin. This is well shown in Fig. 14, where the fibulæ in portions are little more than fibrous bands. Curvature is quite common and the delicate bones are marked by callus at the seats of complete or incomplete fractures of various ages. Epiphyseal separation may lead to obliteration of the cartilaginous lines and arrested longitudinal growth of the shafts, as in the case of the lower ends of the femurs in Fig. 14.

In the **infantile form** all of the bones are slender with thin cortices and remain so whether they become the seat of fractures or not. Healing of fractures occurs in the same way as in the congenital form, and marked absorption in the deeper portion of the periosteal callus and its underlying cortex is frequently seen, giving rise to the formation of the absorption ring of Kienböck. When the fracture is incomplete, the absorption band is limited to the injured side of the bone. Curvature of the weight bearing bones is the usual result when they are the seat of complete or incomplete fractures. Continued apposition on the concave side with absorption on the convex side so displaces the medullary canal that it may come to the surface at the point of greatest convexity, as shown in Fig. 18. Curvature may be so marked as to arouse the suspicion of an associated osteomalacia, but histologic examination of such areas have been negative for osteoid tissue, except in the case reported by Axhausen, where he concluded that osteomalacia formed a part of the picture. Periosteal callus is more completely absorbed than endosteal and transverse bands of the latter may mark the seats of fractures years after their occurrence.

Late cases usually show a frail condition of the shafts of the bones, some of which are marked by old callus at the seats of fracture. Deformities are usually present, but curvatures such as are seen in the cases developing earlier in life do not result. Bones of normal size and density may be predisposed to fracture because of the abnormal distribution of trabeculae and the absence of their normal mathematical arrangement to meet the stress and strain to which they are subjected.

Microscopic Pathology.—Microscopic examination shows a growth deficiency on the part of the osteoblasts which is evident in both transverse and longitudinal bone formation.

The vault of the cranium shows a connective tissue membrane poor in cells, and its plaques of thin bone consist of narrow, irregularly arranged trabeculæ. About the periphery they have a covering of epithelial-like polygonal osteoblasts. There is no differentiation into tabulæ and diploë.

In the long bones the fibrous layer of the periosteum is of normal thickness, but the cambium layer is imperfectly developed throughout, very few osteoblasts being seen along the surface of the imperfectly formed cortex. Lovett and Nichols have studied carefully the microscopic appearance of the thin, irregular cortex. The normal system of haversian canals is lacking and bony lamellæ running parallel to the long axis of the shaft are scarcely to be seen. The trabeculæ run irregularly and cancellous spaces of considerable size may be found in the cortical region, giving it a spongy structure. In the metaphyseal regions cancellous bone is small in amount and is replaced by marrow. Perforations of the cortex are not infrequent with direct communications between the medullary and subperiosteal regions. There is evidence of retarded enchondral ossification at the ends of the shafts. The cartilage of the epiphyses is normal in appearance, but the zones of cartilaginous growth and of calcification in the cartilaginous epiphyseal lines show less activity than normal. The columns of hyaline cartilage are narrower and poorer in cells, containing not more than half the normal number. The zone of calcification is almost entirely lacking. The zone of ossification shows a scanty number of trabeculæ with comparatively small amount of osteoid substance. The trabeculæ are short and thin and do not have the normal longitudinal arrangement, but run irregularly. There is a moderate preponderance of marrow substance. Away from the epiphyseal line there is a rapid disappearance of cartilaginous remnants. Osteoclasts are numerous, while osteoblasts are present in reduced numbers. The bone marrow in general is anemic and may be fibrous in much of its extent. According to Buday, lymphoid marrow is abundant toward the middle of the diaphysis. Much interest has been aroused by the periosteal bands found occasionally in the junction of epiphysis and diaphysis, which may extend from the periosteum and dip into the substance of the epiphyseal cartilage. Lindemann describes them as composed of fibrous connective tissue accompanied by long vessels, and in some instances their presence may greatly distort the cartilaginous columns. The formation is usually more marked on one side than on the other, in which event the epiphyseal line is sloping. It bears considerable resemblance to the bands seen in chondrodystrophy.

Histologically the callus formed at the seat of fractures differs considerably in structure from that formed in normal bones. Periosteal callus is more extensive than endosteal, and considerable metaplasia of the fibrous into cartilaginous tissue occurs preliminary to its ossification, but extensive absorptive changes frequently go on in the deeper portions of the new bony callus and the underlying portions of cortex, leading to the formation of irregular cavities. This may be so marked as to lead to refracture. Cartilage may accumulate in excess, especially in the case of fractures of the ribs, where it may comprise nearly all of the periosteal callus. Extensive absorption may also occur in the osteoid endosteal callus before it is transformed into bone.

Roentgenologic Appearance.—The roentgenologic appearance is usually quite distinct. It is of assistance in distinquishing the congenital form from chondrodystrophia fetalis and the infantile form from severe rickets with its resultant fractures. In general all of the bones show reduction in density. The epiphyses and short bones are normal in outline. The shafts are reduced in diameter and their cortices thin. The markings of numerous fractures are seen and absorption bands may be present in the healing fractures. Periosteal apposition and fraying of the epiphyseal regions as seen in rickets are not present.

Diagnosis.—The congenital form must be distinguished from chondrodystrophia fetalis, with which it was formerly grouped under the heading of fetal rickets. The feature of greatest similarity is the shortening of the extremities in the two conditions, but this is more marked in chondrodystrophy. The fingers are short and stubby in chondrodystrophy, while in osteogenesis imperfecta they are delicate. In chondrodystrophy the head is large, the cranium well formed and the nose is usually saddle-shaped. In osteogenesis imperfecta the head is frequently small, the cranium soft and the face normal in all its features. Fractures are present in osteogenesis imperfecta, which is never the case in chondrodystrophy.

The infantile form is distinguished from severe rickets by the absence of the usual symptoms and signs of the latter condition, aside from the presence of fractures. The roentgenologic findings of absorption bands and straight epiphyseal lines without extra reduction of density and expansion of the metaphyses make the differentiation comparatively easy. Occasionally the two processes are found associated. Where curvatures are marked, it may be confused with juvenile osteomalacia, but the history, the occurrence of fractures without curvatures and absence of evidences of absorption in the metaphyses are against osteomalacia.

Treatment.—Various remedies have been employed in an endeavor to improve osteogenesis and to do away with the abnormal fragility of the bones. Extracts of various glands of internal secretion, cod-liver oil and lime salts have been tried without avail. Phosphorus has been tried repeatedly and usually without success. The result obtained in the patient in Fig. 13 shows that when administered in proper doses for a sufficient length of time its action may be decidedly beneficial. Figure 14 shows the condition of the leg bones at the age of eight, before treatment was started. One two hundreth of a grain of phosphorus was given three times a day for two years, when roentgenograms showed marked stimulation of both longitudinal and transverse bone growth. The drug was continued irregularly during the next two years in $\frac{1}{50}$ gr. doses, when roentgenograms (Figs. 16–17) again showed the same effect. Heavy shadows are shown at the growing ends of the shafts of the leg bones, especially at their lower ends, and there is an increase in diameter as well as in thickness of cortex of both tibia and fibula. The patient, who weighed 25 lb. at the beginning of treatment, increased 12 lb. during the four years, which was a more rapid gain in weight than he had shown before. Joachimsthal observed rapid improvement from phosphorus administration in a child of three months. Within three months all fractures were healed and the bones had assumed practically normal appearance. These experiences would indicate that the drug should be given a more thorough trial than it has heretofore received.

Great care should be taken in the handling of these infants and children to avoid the production of fractures. When fractures occur, they should be treated in the usual way and should not be neglected, in order to guard against the development of marked deformities. It should be remembered that healing occurs in normal time and that pseudarthrosis is rare.

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CHONDRODYSTROPHIA FETALIS

This term is used to designate the commonest form of congenital disturbance of bone growth in which the process of bone formation through cartilage is affected, resulting in disproportioned dwarfism with a large head and unusually short but strong extremities.

The condition is described in earlier medical writings and depicted in the drawings and paintings of the dwarfs of the European courts. Its separation from cretinism is due to the work of Parrot, who in 1878 gave it the name of achondroplasia and first observed that the disturbance is in the cartilage. At first he considered that it was found only in the stillborn and in infants dying soon after delivery, but he subsequently recognized the condition in later life. Kaufmann was the first to describe in detail the different changes that occur in the cartilage and gave it the more appropriate name of chondrodystrophia fetalis, although Parrot recognized that the lesion is a dystrophy instead of an aplasia of the cartilage, as his designation implies.

Etiology.—The condition is always congenital in origin, but little is definitely known of the underlying factors that bring about the growth disturbance. Heredity is of some importance. Several members of a family may be affected, and the disease has been known to run through as many as six generations. The author has seen a chondrodystrophic brother and sister whose aunt was affected with mild gigantism. It has occurred in twins in a number of instances. The condition is one of retarded growth from disturbance of osteocartilaginous development. This affects primarily the entire skeleton which is preformed in cartilage, and secondarily the entire organism.

Attempts have been made to attribute the disease to changes in the thyroid gland. Virchow and Grawitz considered it a form of cretinism and this contention was supported by the post-mortem findings of congenital struma by Moro and Stoeltzner. However, true congenital cretinism has not been reported, and the mental and skeletal developments in the two cases are quite distinct. Because of the gross similarity in the skin changes in the two conditions, it has been considered as allied to myxedema, but Sumita has shown that the two are quite different. In chondrodystrophia fetalis there is simply an excessive accumulation of subcutaneous fat with slight perivascular infiltration and edema of the cutis. None of the other ductless glands have been so far held responsible for the condition.

Other common congenital anomalies have been found with slightly greater frequency in chondrodystrophia fetalis. Hindrance of growth through external mechanical influences, such as pressure of the fetus by abnormal amounts of amniotic fluid or amniotic anomalies and adhesions, cannot be regarded as factors of importance.

Physical Characteristics.—The majority of chondrodystrophics are stillborn, either prematurely or at term. Most of those born living die in the first few days or weeks. Those surviving this period are ordinarily resistant and may live to a ripe old age. The physical appearance at whatever period seen is very characteristic.

The stillborn fetus has an abnormally large head and short, stubby extremities in comparison with the trunk. The skin over the entire body is loose and in folds, especially about the joints, neck and chin and wherever normal creases are to be found. The head has the appearance of being hydrocephalic, the cranium is large and high with a prominent brow. Its bones are ossified, but the sutures of the fontanelles are wide open, even in full-term fetuses. The face is relatively small and presents a stupid appearance, resembling that of a cretin. One of its most striking alterations is the nasal deformity. The bridge of the nose is usually markedly retracted, the tip rounded and up-turned, the alæ thickened and the orifices narrow. The skin of the face is thick, which increases the folds about the eyes, ears, nose, mouth and chin and produces a puffiness of the eyes and cheeks. There is eversion of the lips.

The trunk has fewer changes. It is smaller than normal. The neck is short and thick, with deep folds in the skin. The thorax is somewhat funnel-shaped and the abdomen prominent, giving the trunk somewhat of a spindle shape. Lumbar lordosis is usually present, making a protrusion of the buttocks. The external genitalia have no anomalies.

Pathology.—Changes in the soft parts are almost wholly secondary to the skeletal alterations. The skin is loose and folded and appears too large for the body. The subcutaneous fat is increased. Occasionally there may be very marked obesity in the surviving cases. McCallum reported the autopsy of a 75 year old woman who was 45 in. tall and weighed 150 lb. Muscles and tendons are short but well developed. The thoracic and abdominal viscera are unchanged aside from slight alterations in shape. The ductless glands are unaltered, except for the occasional finding of adenoma of the thyroid.

Gross Changes in the Skeleton.—These have been described most completely at birth, because of the large number of skeletons of new-born infants that have come to examination. They are most striking in the bones of the extremities. In general the shaft of the bone is very short and slightly reduced in diameter, but the cortex is dense and strong. A certain amount of curvature is common. It possesses a good periosteal covering. The ends of the shaft expand to meet the relatively much larger cartilaginous epiphyses, which usually present considerable alteration in shape. The epiphysis is generally normal in consistency, but may be abnormally soft and vascular. When sawed open, the cortex of the shaft is well formed, but the medullary canal is very imperfectly established, the interior containing much spongy bone. The epiphyses are devoid of a center of ossification, aside from that of the lower end of the femur, which may be present at birth. Grossly there is little abnormal to be seen at the junction of shaft and epiphysis, except occasionally the periosteal bands which bend around the limits of the shaft and extend inwards to a variable depth between metaphysis and epiphysis.

Certain of the extremity bones present special features. The femur usually has very large and deformed epiphyses. The femoral neck shows a coxa vara and the head is usually small and flattened on top. The shaft is frequently curved and the condyles rotated slightly outward. The tibia and fibula are more nearly of equal length than in the normal and are frequently curved outward. The humeral shaft is short and the epiphyses much distorted. The head shows different degrees of development and a varus position may exist. The forearm bones are usually curved outward and the hand may be in slight radial flexion. The fingers and toes are short, and the hand especially has a quadrilateral outline. The carpal and tarsal bones show little alteration as to size or form.

The skull is abnormal in shape and the face deformed as a result of the unequal development of the different cranial bones. The bones of the vault, which are preformed in membrane, show no retardation in growth, but the general form of the vault is changed, being hydrocephalic in outline. This is largely secondary to the disturbed growth of the bones of the base, that are preformed in cartilage and affected with the same regularity as are the bones of the trunk and extremities. Mild hydrocephalus may be present and spontaneous cure sometimes results in surviving cases.

The changes at the base of the skull have received a good deal of attention in reference to the retracted bridge of the nose. The base of the skull is formed by a group of bones to which Virchow gave the name of os tribasilare. It consists of the two sphenoid bones and the basilar portion of the occipital bone, all of which are preformed in cartilage and separated from each other by synchondroses. The cartilaginous disc between the anterior and posterior sphenoidal bones remains completely open for only a short time after birth, but the spheno-occipital synchondrosis first begins to close around the thirteenth year and the process is not complete until the eighteenth or twentieth year. A study of the base of the skull in chondrodystrophia shows it to be considerably shorter than normal, which according to Frangenheim may be due to the following conditions:

1. Synostosis of the Tribasilare.—This was at first considered by His, Marchand, Paltauf and others to be the sole cause of the saddle nose. Kaufmann has shown that there are two groups of cases presenting retracted nose: One with retraction of the root only, in which premature synostosis is not always found, and the other with flattening of the entire nose, in which the tribasilare is usually cartilaginous, soft and irregularly formed. In this group the flat nose and facial changes are not determined by the configuration of the sphenoid, but by a defective growth of the cartilaginous skeleton (nasal and supramaxillary parts according to Kaufmann) situated in front of the tribasilare.

2. Open sutures with small bones due to retarded growth.

3. An almost completely cartilaginous condition of the base of the skull with only a trivial amount of ossification. These two latter conditions are not infrequently found, especially where a flattened condition of the nose is present. The sphenoidal bone is steeper than normal and in some cases may form almost a straight line with the cervical spine. This usual shortening of the base of the skull does not interfere with the growth of the brain, although its form is somewhat altered thereby.

The spine shows changes analogous to those of the long bones. The only alteration in curvature consists in an increase of the lumbar lordosis. The bodies of the vertebræ are relatively small. As a result of the retarded ossification, Breus and Kolisko and Sumita have found at birth in a number of cases a synostosis of the centers of ossification in the body and the lateral masses, forming a complete bony ring. In other cases synostosis does not occur and the centers of ossification remain small. When synostosis occurs the spinal canal remains small and the spinal cord is compressed, which may be responsible for the great frequency of death either before or soon after birth. The clavicle is practically unaffected, due no doubt to the fact that it is preformed in membrane. The ribs show little change except at the costo-chondral junction, where there is a well-marked rosary, bulging chiefly to the inside. On longitudinal section this is found to be due to expansion of the end of the bone and in some cases to cup-shaped overgrowth of the bone around the end of the cartilage. Occasionally the rosary is the result of enlargement of the end of the cartilage. The pelvis has been described in detail by Sumita. In general it is small and resembles the contracted rachitic pelvis. The iliac wings are little curved and the crests heavy. The pelvic inlet is markedly stenosed and flattened anteroposteriorly. The sacrum is almost horizontally placed, partly as a result of the lumbar lordosis, and its promontory projects unduly. The pelvic cavity is relatively large and broadens from above downward in its anteroposterior diameter, due to the abnormal rotation of the sacrum.

Microscopic Pathology.—The disturbances in bone formation are seen in the regions where enchondral ossification occurs, namely, through the epiphyseal lines of the ends of the shaft and in the centers of ossification of the epiphyses and short bones. The lesion is in the cartilage, which structure does not prepare the way for the normal amount of bone growth. Kaufmann has divided the changes that may occur in the cartilage into three forms:

1. Malacic chondrodystrophy, in which the cartilage is soft.
2. Hypoplastic chondrodystrophy.
3. Hyperplastic chondrodystrophy.

This distinction is of little importance and is to be made out mainly in the bones at the base of the skull, which are the only ones in which the malacic form occurs. The changes in the trunk and extremity bones are nearly all of the hypoplastic form. In this form the mass of cartilage in the epiphysis is composed of irregularly distributed small spindle-shaped cells with granulated nuclei, with more intercellular substance and less ordered arrangement than in the normal. Large numbers of blood-vessels may be found throughout the cartilage and it usually has a peripheral covering of connective tissue. The only regions in which the cells show any tendency to columnar arrangement are those bordering the periphery of the centers of ossification and the growing ends of the shaft. Normally the cartilage cells at the ends of the shafts are arranged in long, parallel, closely approximated columns. In chondrodystrophic epiphyses there is usually only a suggestion of columnar arrangement about the center of ossification but the cells are more spherical and larger than in the rest of the cartilage. At the end of the shaft the columnar arrangement may be more marked, but the columns are short, irregular and separated from each other by a wide intercellular substance.

Activity in the zone of cartilage growth is so slight that little change in the character and number of its nuclei can be made out. The zone of calcification is extremely scanty and irregular, and islands are frequently included in the columns of spongy bone which grow in from the zone of ossification and replace it.

The condition of the diaphysis shows wide variations. In some cases the periosteal bone formation has replaced the tissue of the shaft so that the central spongy bone has almost disappeared; in other cases the spongy enchondral bone is retained and the marrow cavity is lacking, but there are many large marrow spaces bounded by irregular, delicate trabeculæ. Toward the epiphyseal border the marrow spaces become narrower; jagged projections in the zone of ossification are occasioned by the marrow spaces breaking into the cavities containing the blood-vessels. These marrow spaces open also into the calcified and hyaline cartilaginous substance; bone marrow giant cells, lying in lacunæ, play a rôle in their formation. A delicate margin of bone lines the hollow spaces. The trabeculæ bounding the marrow spaces are blue in the center (calcified cartilage—Marchand, Kaufmann), are constructed in strata at the periphery and possess a covering of osteoblasts.

In a case reported by v. Franqué, the diaphysis in the regions of greatest curvature consisted of compact bony substance, so that the marrow cavity was lacking entirely in the humerus and ulna and partly in the radius. These solid areas are of exclusively periosteal origin, and are sometimes chiefly formed from the concave side of the bone in question, where the cambium layer of periosteum shows marked thickening.

The bone marrow is normal in appearance.

The periosteal bands of white fibrous connective tissue which dip inward at the junction of epiphysis and diaphysis are a characteristic finding in chondrodystrophia fetalis and aid greatly in distinguishing it from other forms of disturbed bone growth. Their method of formation is not well understood, but they probably arise from an infolding of the periosteum due to the disproportion of enchondral and periosteal growth, the latter being the more active. They are usually found at both epiphyses of a shaft and extend inward to varying depths, where they gradually lose themselves in the cartilaginous substance, but rarely unite to divide the epiphyseal cartilage from the diaphysis. They greatly interfere with longitudinal growth of the shaft, and when more marked or present only on one side produce a curvature with the convexity of the shaft in that direction.

The centers of ossification in the bodies of the vertebræ are surrounded by a broader zone of cartilage growth with more columnar arrangement than is seen in the centers of ossification of the epiphyses of the long bones. These centers of ossification have jagged outlines and vascular marrow spaces which penetrate into the zone of calcification of the cartilage and in some instances are connected with vessels in the cartilage. The vertebræ may show findings which suggest the time of onset of the trouble. In a case of Kaufmann's, the chorda dorsalis was seen as a continuous strand in the region of the upper thoracic vertebræ. Since the bony center of the vertebral body arises near and posterior to the chorda, later surrounding it, the onset of the disease in this case must have been before or during the second fetal month, at which time the enclosure takes place. In other cases with longer bones the onset of the trouble is in a later period of intra-uterine life. Consequently, there is no set period for the onset of the process.

The tribasilar bone of the floor of the skull shows three types of changes. In the malacic form the cartilage is mainly unossified and there is an absence of fusion of the centers of ossification. The periphery of the center of ossification is irregular and the cells of the adjacent cartilage have a disordered arrangement. The remaining cartilage is more homogeneous and hyaline. The number of blood-vessels is greatly increased and areas of softening may be found irregularly distributed. In the rarer hyperplastic form activity in the zone of cartilage surrounding the centers of ossification is great, while in the hypoplastic form it is extremely slight. In these two forms various degrees of fusion of the centers of ossification in the bones are nearly always present, thereby shortening the os tribasilare.

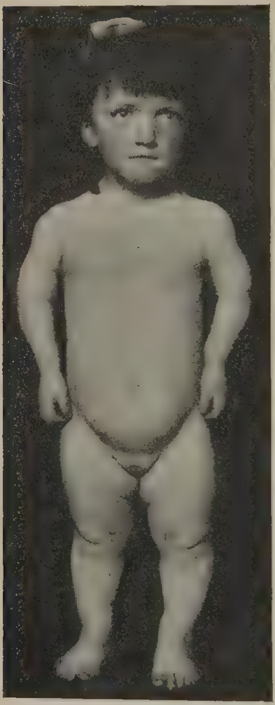

FIG. 20.—Chondrodystrophic dwarf, 6½ years old, 34 inches tall.

Surviving Cases.—Life is not seriously threatened after the first few months are past, and body development proceeds, but at a somewhat slower and more uneven rate than normal. On the other hand, the capacity for mental development is normal. The children learn to walk and talk at the proper age and are ordinarily intelligent, but subsequently are deprived of the opportunities for proper social and mental development because of their physical unfitness for normal work and social activities. The characteristic disproportions at birth persist in later life. The head continues abnormally large in comparison with the trunk and extremities, and the brow prominent. Dandy has recently reported two cases of chondrodystrophy in which moderate hydrocephalus was demonstrated by means of ventriculography. Mentality was impaired in one case, in which spontaneous cure of the hydrocephalus had occurred, as shown by the fact that air injected into the ventricles escaped through the foramina of Luschka and Magendie into the subarachnoidal space. He concludes that the size of the head and therefore the grade of hydrocephalus seem to be proportionate to the severity of the dwarf phenomena in chondrodystrophy. The bridge of the nose remains sunken, the face flat, and the chin becomes unduly protruding (Fig. 1). The trunk is short, but the extremities are disproportionately shorter. The buttocks are very prominent, due to the lumbar lordosis. The finger tips usually reach just below the iliac crests. The arms are slightly curved and relatively shorter than the forearms. In the erect position the elbows usually stand out from the body, due to the undeveloped humeral head. The hands are short, the fingers stubby and nearly equal in length. Occasionally, the third finger

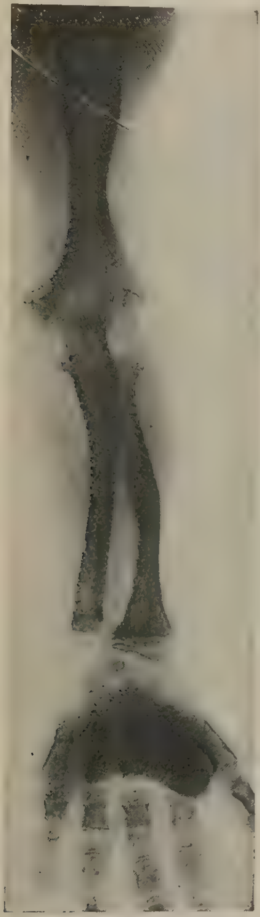

Fig. 21.—Upper extremity of case in Fig. 20.

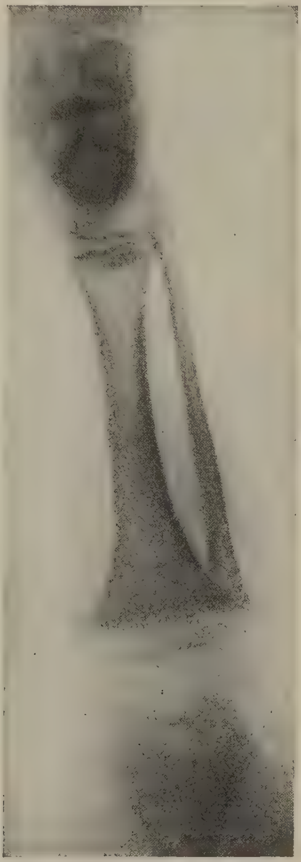

Fig. 22.—Leg bones of case in Fig. 20.

deviates to the ulnar side at the first phalangeal joint, giving the hand a forked appearance. The thighs are very short and fat, more so than the legs. The feet are also stubby. The muscles are well developed and many chondrodystrophics function in circuses as acrobats and clowns. However, the undeveloped head of the humerus may limit abduction of the shoulder and the coxa vara abduction of the hip, producing a waddling gait. There is no special predisposition to fractures. Full-grown chondrodystrophic dwarfs usually reach the height of from 40 to 50 in.

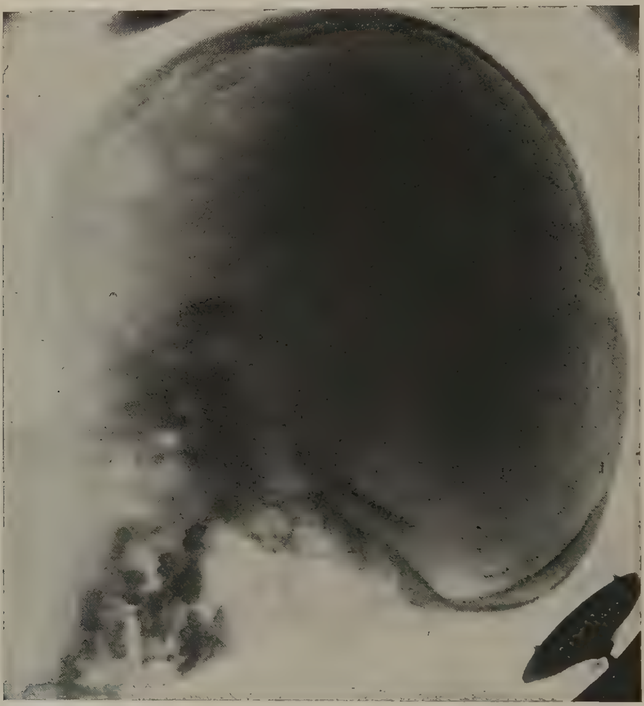

FIG. 23.—Skull of case in Fig. 20. Tribasilar synostosis.

Skeletal Findings.—A number of growing and adult skeletons have come to gross and microscopic examination and Roentgen-ray studies have thrown much light upon the form and development of the bones of chondrodystrophics of different ages. In general the bones are short, small and somewhat malformed. Epiphyses and short bones show considerable change. The centers of ossification of the epiphyses appear generally at the normal time. They enlarge gradually and usually fuse with the shaft at the proper age, but frequently their appearance, ossification and fusion are delayed, sometimes for many years, as shown by the observation of Marie, Joachimsthal and

Frangenheim. Thus ossification in the caprus and hand epiphyses may be no farther advanced at the age of 10 than in a normal child at the second year. On the other hand, Kassowitz described cases with premature ossification and synostosis in the extremity bones. The small humeral head, the coxa vara and large deformed trochanters and the distorted epiphyses about the knees, ankles, and elbows are well-marked features.

The short and frequently curved shafts may be abnormally prominent at the points of muscle insertion and their ends are frequently distorted at the lines of junction with the epiphyses. A characteristic finding is the abnormally long fibula, the upper epiphysis of which reaches the level of the knee joint. Its prominent lower end may produce inversion of the foot. The base of the skull in all surviving cases shows shortening with premature synostosis and a reduction in the angle of the base (Fig. 23). All of these changes have been extensively studied at every age by means of roentgenograms. The malformed epiphyses and ends of the shafts, the irregular epiphyseal lines and the delay and irregularity of ossification in the short bones and epiphyses make the diagnosis of chondrodystrophy from roentgenograms of the joint regions comparatively certain. In Fig. 21 of a six and one-half year old chondrodystrophic, the centers of ossification of the external condyle, internal epicondyle and radial head are present, but smaller than normal. At the wrist the carpal centers are nearly normal, but the forearm epiphyses are undersized. All epiphyseal lines at the ends of shafts are excessively uneven and jagged. Figure 22 shows similar changes in the lower extremity, but most marked in the lower epiphysis of the femur, the inferior border of which is extremely distorted. Figure 24 of a 29 year old dwarf shows the classical features of chondrodystrophy. Figure 25 shows especially well the altered humeral head and the radial curve with apposition on its concave and absorption on its convex sides. Figure 26 shows the distorted epiphyses, especially of the external condyle of the femur, and the long fibula with its upper end reaching the level of the knee joint. The external malleolus extends lower than normal and the ankle joint is altered thereby.

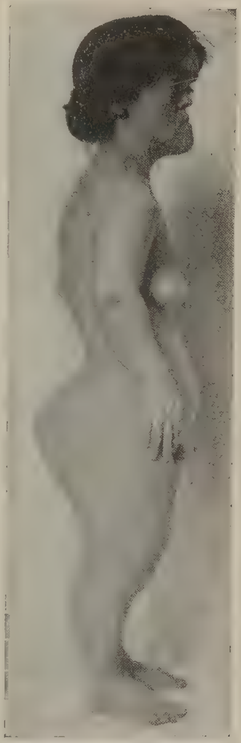
FIG. 24.—Chondrodystrophic dwarf, 29 years old, 44 inches tall.

Treatment.—Since the condition is a congenital disturbance, nothing can be done to modify its course. Phosphorus, which influences especially longitudinal growth of the shafts through enchondral bone formation, has not been tried, but it is unlikely that it would have a beneficial result since its action is upon the zone of ossification and not on the zones of cartilage growth and calcification, which are at fault in chondrodystrophy. Corrective orthopedic measures are occasionally indicated in case of bad curvatures of the limbs. Thus Joachimsthal and Hoeftman osteotomized a number of cases with permanent satisfactory results. Barth had a non-union of the femur in one case. Cramer saw recurrence of the deformity after osteotomy and correction. Because of the tendency for deformities to increase during the growing period, corrective operations should be resorted to with a good deal of caution before adult life is reached.

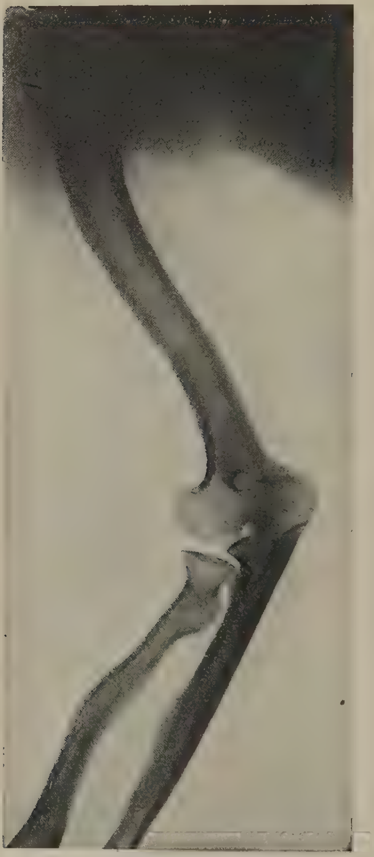

Fig. 25.—Arm and forearm of case in Fig. 24.

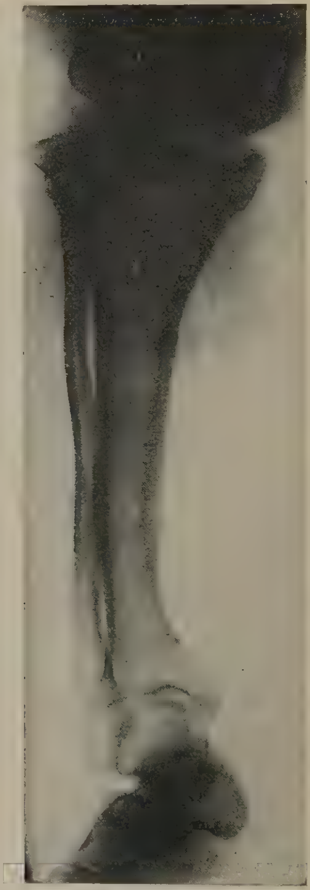

Fig. 26.—Leg bones of case in Fig. 24.

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MULTIPLE CARTILAGINOUS EXOSTOSES

This is a disturbance of enchondral bone growth beginning in childhood and characterized by uneven shortness of the long bones, expansion of the ends of the shafts and the presence of multiple, cartilage capped exostoses springing from the ends of the shafts of the long bones and from the bodies of the bones of the trunk.

Etiology.—The disease has a marked hereditary tendency, one or more members of a family being affected, frequently extending through several generations. It affects both sexes and is transmitted by either sex, but is found much more frequently in the male. One or two generations may be skipped in the transmission of the disease. It is comparatively rare to find isolated cases, and numerous family trees have been worked out showing its transmission. The disturbance is rarely manifested at birth, although the tendency to its development is undoubtedly inherited. The nature of the disease is unknown, but it belongs to the growth disturbances rather than to true tumor formations. That this is so is shown especially by the fact that bones showing the greatest degree of shortening may be entirely free from tumors. The disorder is of the cartilage rather than of the bone, as is shown by pathological examination, which reveals a variety of cartilaginous changes. Exostoses and enchondromas are sometimes present in the same patient. Numerous contributary causes have been mentioned, as rickets and disturbances of internal secretion, particularly of the thyroid, but they are only occasional and incidental occurrences. Very frequently a single exostosis is attributed by the patient to previous traumatism, especially if it is the first to be noticed, but injury probably never plays a part in the development of multiple tumors. Virchow, Fischer and Eberth reported a recurring febrile condition associated with the development of the exostoses, but this has not been substantiated by extensive subsequent observations.

Clinical Manifestations.—Nearly all of these children are apparently normal at birth, although there are several reported cases in which tumors were noted at or soon after birth. Usually the first change noticed is the presence of hard tumors near the ends of some of the long bones of the extremities between the fifth and the eighth years. Shortness of the fingers and forearms and ulnar flexion at the wrists may be an early finding. With increse in age the tumors enlarge, new ones appear and the deformities increase in severity until full growth of the skeleton is reached. The picture of the condition is well marked during adolescence and is as follows:

The individual is usually short of stature, but never to the extent of dwarfism. On the other hand, cases of more than average height may be seen. This arrest in longitudinal growth is disproportioned. The upper half of the body is longer than the lower and the lower extremities relatively shorter than the upper. The head is normal, as bone preformed in membrane is unaffected, and the base of the skull, which is of cartilaginous origin, is rarely affected. The forearms are frequently deformed and the hands in ulnar flexion. This condition is due to shortness of the lower end of the ulna, which is usually more marked on one side than on the other. When extreme, it may be associated with a resultant dislocation of the head of the radius. A corresponding, but usually less marked, shortness of the fibula is often met with, which gives rise to eversion of the foot with obliquity of the ankle joint and prominence of the internal malleolus. Enlargements produced by the tumors may be seen in connection with any of the bones of the trunk or extremities. They are most noticeable about the ends of the shafts of the long bones, especially about the knee, ankle, upper end of the humerus, wrist and in the metacarpals and phalanges of the hands. The clavicle, scapula and ribs are frequently involved, and tumors along the crest of the ilium are not uncommon. Visible or palpable tumors in connection with the spine are very rare.

The enlargements vary from scarcely visible masses to those as large as a child's head. Usually they are irregular, oval shaped bulgings about the ends of the shafts, but round and pedunculated masses may spring especially from the hand bones and tibia. On palpation they are of bony consistency, although occasionally a thick cartilaginous cap or bursal covering may give an elastic or fluctuating feeling to the extremity of the tumor. Usually many small masses can be felt that are not to be made out on inspection. Diffuse expansion of the ends of the shafts may be elicited on palpation, particularly of the upper end of the tibia and humerus and of the lower end of the femur (Fig. 27). The tumors are painless and rarely produce symptoms except as they interfere with motion, and since the epiphyses are free this tendency is not great. Large tumors about the hip and shoulder may restrict motion in these joints, and tumors from the lateral portion of the ribs may considerably displace the arm, as in a case reported by Frangenheim. Dislocation of the head of the radius, associated with tumors about the elbow may interfere with motion in that joint, and pronation and supination may be reduced in varying degrees by the tumors of the radius and ulna. Genu varum may be sufficiently marked in one limb to produce a limp, and difficulty in walking may result from the eversion of the foot and prominence of the internal malleolus, which are secondary to the shortened fibula. Large tumors of the hands may greatly interfere with their use. This is especially apt to be the case when metacarpal phalangeal enchondromata are associated with the exostoses.

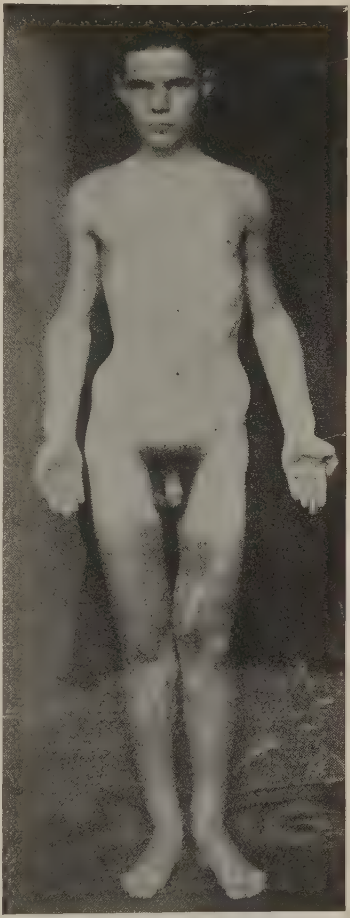

FIG. 27.—Multiple cartilaginous exostoses, seventeen year old boy.

Pathology.—The disturbed enchondral ossification results in arrested growth and deformities of the shafts of the extremity bones and the bodies of the short and irregular bones and tumor formations in their peripheral portions. The epiphyses are rarely involved, except secondarily as they are distorted by the disturbances in the shaft. Their centers of ossification appear at the right time and ossification proceeds normally. Tumors practically never take origin in the epiphyses. The extent of involvement and number of bones involved are extremely variable. While any bone preformed in cartilage may be affected, some are more prone to involvement than others and all bones are practically never involved in one case. Different types of change are commoner in some bones than others. Thus tumor formation is most frequent in the femur, while marked shortening is most frequent in the ulna. Arrested longitudinal growth in the long bones is disproportioned. While all of the bones are usually slightly shorter than normal, some are more involved than others and homologous bones are not equally affected. The ulnæ and finger bones show the most marked degree of involvement in this respect. Shortness is the result of defective growth and not secondary to the presence of tumors, because while there is some direct relation between the number and extent of tumors and the amount of shortening, as pointed out by Bessel-Hagen, occasionally the most marked arrestment of longitudinal growth occurs in the entire absence of tumor formation. Histologic examination of the epiphyseal lines fails to show any appreciable disturbance in the cartilage, such as exists in chondrodystrophia fetalis, to which this condition bears slight resemblance. Expansion of the ends of the shaft is more closely allied with the presence of tumors in this region. It is seen especially in the ends of the long bones from which most rapid growth takes place, as the upper end of the humerus and lower end of the femur, but may occur in any tumor bearing region. The expansion may extend from the epiphysis several inches back on the shaft. It is inclined to be cylindrical, but is usually more marked in the segments that are the seat of tumor. The bone has a blown up appearance, as the cortex in the expanded region is thinned out and streaked with irregular trabeculæ, the heavier ones of which may stand out as longitudinal lines in roentgenograms (Fig. 28). Interior trabeculæ are fewer and atypically arranged, leaving large marrow spaces that are generally occupied by fat. Distortion of the short and irregular bones of the skeleton is less frequent than

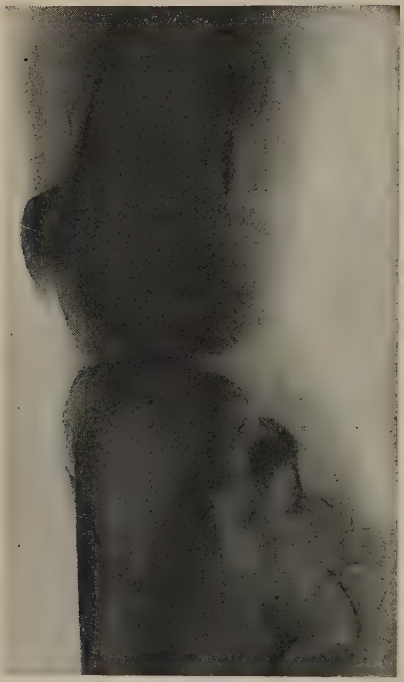
FIG. 28.—Roentgenogram of left knee of case in Fig. 27.

that of the long bones. Occasionally there is deformity of the pelvis and very rarely of the spine. Tumor formation is the most striking feature of the disorder. In general the tumors spring from the sides of the growing bones in close proximity to the epiphyseal lines.

The most commonly accepted theory as to their origin is that they arise from peripheral portions of an abnormal cartilaginous epiphyseal line, which do not ossify but deviate and form islands of cartilage along the surface of the metaphysis. These subsequently grow at right angles to the long axis of the shaft and form bony tumors. The cause of this deviation and isolation of islands of the cartilage has been variously attributed to the traction of muscles and membranes upon their seats of insertion and to traction of periosteum at the periphery of the epiphyseal line as the epiphysis is pushed ahead by the growing shaft. This view is supported by the location of some of the tumors at the seat of muscle attachment, as the insertion of the deltoid, and at the insertion of the interosseous ligament to the lower end of the radius, where spur formation is an almost constant occurrence. Another possible origin is that pointed out by E. Müller, who found, in a case of multiple cartilaginous exostosis in a man dying at the age of 26, numerous islands of cartilage in the periosteum of the ends of the shafts. From this he concluded that a constitutional anomaly exists in the periosteum and perichondrium leading to their metaplasia into cartilage, from which tumors may take origin.

The tumors at first are small and oval shaped and located near the ends of the shafts, but as growth continues those which arise early come to lie at a considerable distance from the epiphysis.

Many of the tumors persist as oval shaped elevations from the cortex; others become hemi-spherical; still others become pedunculated and grow usually away from the epiphysis and parallel with the shafts, forming spurs which extend into the surrounding muscles. The surface of the exostoses varies from smooth and rounded to extremely irregular. Longitudinal section through the end of the bone and the exostosis presents the following picture: The expanded cortex of the shaft is continuous with the base of the exostosis and cortex is absent in the region of the insertion of the tumor, so that medullary cavity and cancellous bone of the end of the shaft extend directly out into the exostosis (Fig. 29). Longitudinal section through the tumor presents the following appearance: Along its surface is an irregular fibrous covering, which is continuous with the periosteum at the base. Beneath this and capping the bony portion is a layer of cartilage which is extremely variable in its extent. In some cases, particularly where the tumor is spherical, it covers the entire surface, but in most cases it is present in various sized islands which cap the irregular wart-like protuberances and in places form large sheets. The layer of cartilage varies greatly in thickness. Small islands may be little thicker than a sheet of paper, while large tumors may be composed largely of cartilage, which is as much as an inch thick in some places. The cartilaginous surface is usually smooth; where the cartilage is extremely thick, it is sometimes lobulated. The peripheral layer is composed of hyaline cartilage, which is usually the thicker, while the deeper layer consists of calcified cartilage and is clearly visible as a yellowish-white zone. Occasionally, this zone is very thick and extends deep into the underlying bone, with the peripheral elements of which it may be intermixed, giving it a chalky and sometimes crumbly nature. Roentgenologically this layer may cast a much heavier and less even shadow than the underlying cancellous bone and can frequently be differentiated from the latter because of its dense shadow. The interior of the exostosis is made up of cancellous bone, the marrow spaces of which are filled with fat and bone marrow similar to that of the end of the shaft.

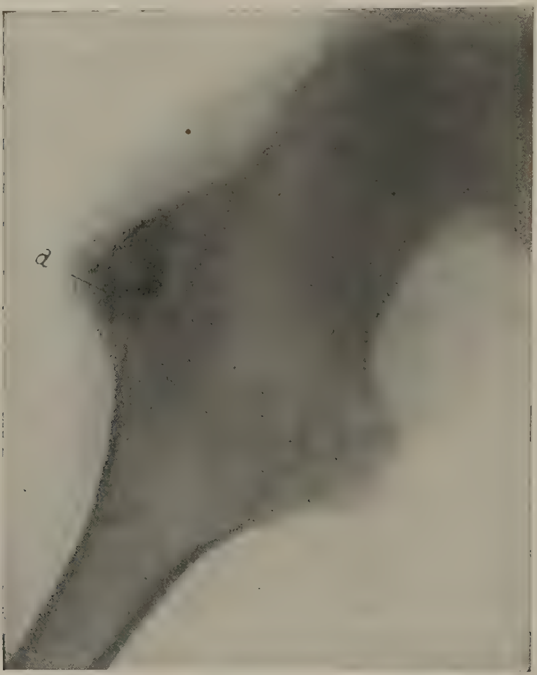

Fig. 29.—Left humerus of case in Fig. 27. (*a*) Dense shadow cast by zone of calcification.

In the case of pedunculated exostoses the pedicle may be long and have the structure of the shaft of a long bone with a periosteum, cortex and medullary cavity. The ends and prominences of such exostoses are always expanded and during the growing period capped by islands of cartilage. Occasionally there is bursa formation over the most prominent portion of an exostosis, which may largely obscure the size of the underlying tumor. These sacs resemble enlarged bursæ in other localities and are filled with a slightly mucous fluid. Their capsules are inserted at the margin of the cartilaginous covering and are in direct connection with the periosteal covering. The inner surface of the bursa is smooth or covered with villi. Occasionally rice bodies form in them, as many as 500 in a case reported by Fehleisen. In the 18 cases of bursa formation collected by Rietus, 10 were about the lower end of the femur. It has been suspected that the origin of the bursa was in the cartilage, but this does not seem to be substantiated by pathologic examination. Rarely in case of a very thick layer of hyaline cartilage about the surface, degeneration and cyst formation occur within its substance, producing a picture which may be confused with bursa formation. Marked overgrowth of the layer of hyaline cartilage may occur, which was described by Haberer as enchondroma formation from the exostosis. Such enchondromas from exostoses have been seen in the plantar region arising from the os calcis.

Fracture of the pedunculated exostosis is not uncommon when about the knee. It usually fails to unite and remains as a loose body in the soft parts, which may produce pain on walking (Fig. 31).

Growth of the exostoses usually ceases with the closure of the epiphyseal lines and cessation of growth of the skeleton; rarely it continues for some years thereafter. On the other hand recession of the tumors has been known to occur after adult life is reached. This is due to disappearance of the cartilaginous covering, as diminution in size of the bony portion does not occur. Figure 34 shows the leg bones of an adult in which the cartilaginous caps on the exostoses had almost entirely disappeared.

Microscopic Structure.—Microscopic examination of the exostosis shows its great similarity in structure to a metaphysis with its cartilaginous epiphyseal line. Growth of the two occurs through enchondral ossification. The cartilaginous cap of the exostosis corresponds to the epiphyseal line and the underlying bone to the metaphysis. The perichondrium covering the cartilaginous cap of the exostosis is composed of white fibrous connective tissue and in the regions where cartilage is absent it covers the bony cortex. A layer of cells similar to the cambium layer of periosteum of the shaft may be present. The outer layer of cartilage is hyaline and the cells in its peripheral portions are evenly distributed throughout the hyaline matrix. In its deeper portion they have an irregular columnar arrangement similar to that of the epiphyseal line. In many cases the columns are extraordinarily irregular and short, while in others they may be much longer than those seen in an epiphyseal line. The former condition is usually seen where the layer of cartilage is thin and growth of the exostosis is exceedingly slight or at a standstill. Degenerative changes even to the extent of cyst formation may occur where the layer is very thick.

Beneath the zone of columnar hyaline cartilage is the zone of calcification, which shows the widest variations according to the size and age of the tumor and the amount of cartilage in its composition.

Roughly, it has the appearance of the zone of calcification in the epiphyseal line, in that it consists of degenerating columns of cells and vacuoles from the zone of hyaline columnar cartilage with a calcified intercolumnar substance. When the cartilage consists of thin islands capping the prominences of an exostosis, the zone of calcification is usually narrow and may be entirely absent where growth has ceased. When the layer of cartilage is more extensive, it may be very thick, comprising in some instances as much as 1 or 2 cm. of the periphery of the tumor. In this case the layer consists largely of amorphous, intercolumnar calcification with the formation of cancellous spaces and scattered bony trabeculæ. Deeper the number of bony trabeculæ increases and gradually the zone of calcification completely merges into the zone of ossification, which consists of cancellous bone similar to that of the metaphysis. Occasionally fair-sized islands of the zone of calcification will be found deep in the cancellous interior. There is absence of any mathematical arrangement of the interior trabeculæ of exostoses, such as is seen in the ends of the shafts.

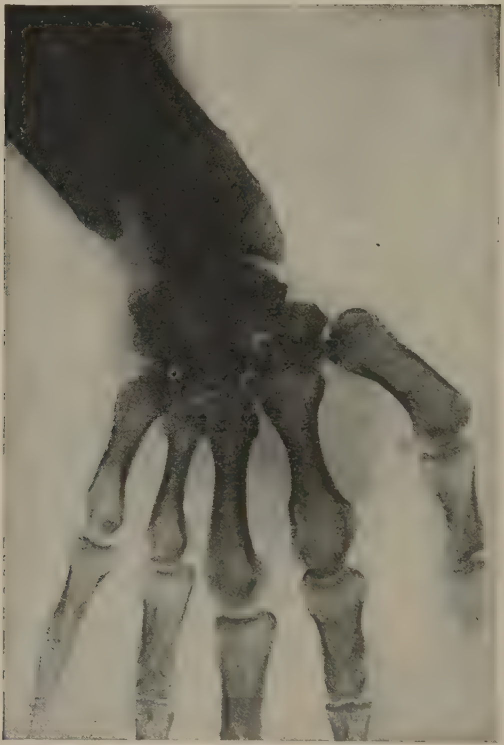

Fig. 30.—Forearm of case in Fig. 27, showing short ulna and exostoses on radius and metacarpals.

Regional Involvement.—The bones of the face and vault of the skull are not involved. The base of the skull has rarely been the seat of an exostosis, usually springing from the clivus of the sphenoid but not reaching sufficient size to produce symptoms. The ribs are very frequently affected. The tumors may be situated anywhere along their course, but most frequently either posteriorly or near the enchondral junction. Enchondromas of the costo-chondral junction may occur in a patient with multiple exostoses. Rib tumors are usually small, but large ones may form and greatly deform the chest. Slight prominences may form on either surface of the sternum. The clavicles usually show thickening and warty excrescences at either end. The margins and spine of the scapula are the seats of predilection for this bone and large tumors may spring from it (Fig. 33). The vertebræ are relatively rarely involved, the superior and inferior margins of their bodies being the commonest seats of the tumors. They may also spring from the lateral mass on either side, but rarely bulge into the spinal canal (Fig. 32). The pelvic bones, especially along their free margins, are frequently the seat of tumors, which are occasionally of very large size. The crest of the ilium is most often involved. The writer saw a large tumor, springing from the posterior part of the ilium, fill the pelvis and produce intestinal obstruction. Occasional deformity and contracture may be the result and Sonnenschein reports ankylosis of the sacro-iliac joints.

The extremity bones show involvement in the following order of frequency, according to Weber: Lower end of femur, lower end of tibia and fibula, upper end of femur, lower end of radius and ulna, upper end of tibia, upper end of humerus, upper end of radius and ulna, lower end of humerus. This leaves out of consideration the hand and foot bones, some of which are involved in practically every case. The lesions are fairly symmetrically distributed, but there may be great variation in the size of the tumors on the two sides. Those of the upper end of the humerus are nearly always sessile and associated with an expanded cortex. They are most commonly attached in the region of the surgical neck, but may extend as low as the deltoid insertion and may bulge in any direction. This is one of the most frequent seats of calcified masses in the periphery of the tumor, casting heavy shadows in the *x*-ray (Fig. 29).

Tumors of the lower end of the humerus are rare. Deformity and limitation of motion in the elbow joint are not uncommon as a result of enlargment and dislocation of the head of the radius. Small exostoses from the coronoid process of the ulna and from the upper end of the radius may add further to limitation of motion. The shaft of the radius is usually curved in case the lower end of the ulna is short. Tumor formation from the lower end of the radius is commoner than from the ulna, despite the shortened condition of the latter. A short spur frequently springs from the radius at the lowest point of insertion of the interosseous ligament. Occasionally there is such extensive tumor formation in this region that radio-ulnar synostosis results. Where the ulna is short, marked deviation of the wrist to that side results, and the plane of the joint may be markedly tilted as a result of the curvature of the lower portion of the radius. Small

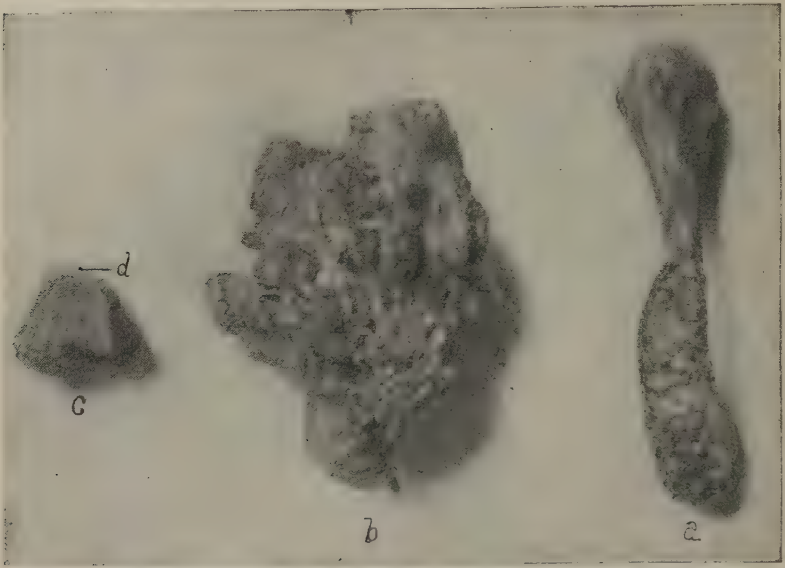

FIG. 31.—Specimens from case in Fig. 27. (*a*) Spur from internal epicondyle of femur. (*b*) Upper end of fibula. (*c*) Lateral exostosis of humerus with (*d*) cartilaginous cap.

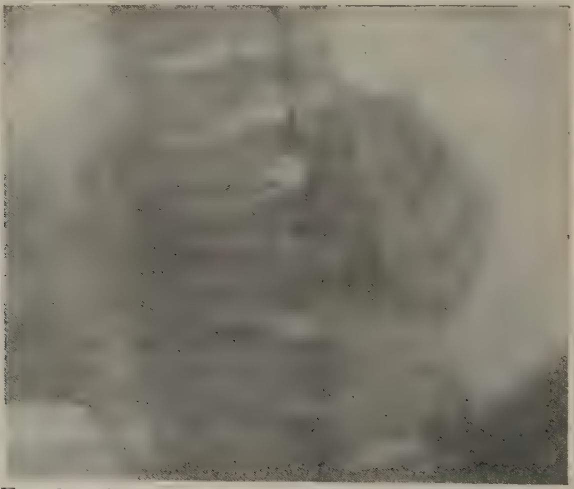

FIG. 32.—Exostosis of cervical spine producing neuritis.

tumors are usually found in connection with the ends of the shafts of some of the metacarpals and phalanges which have epiphyses. They may reach considerable dimensions and interfere with function. Enchondromas may occur with or without the presence of exostoses. Shortening of the affected bones is frequently present (Fig. 30).

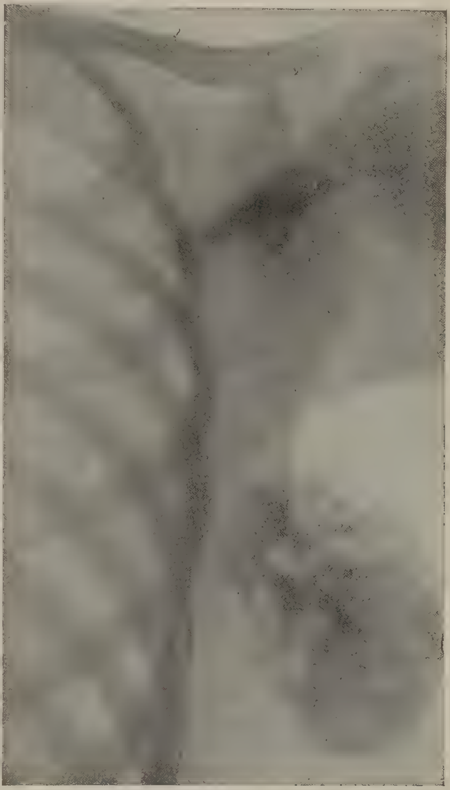

FIG. 33.—Exostosis of scapula.

Tumor formation at the upper extremity of the femur is usually in the trochanteric and subtrochanteric regions and associated with expansion of the metaphysis. Large masses consisting mainly of thick layers of hyaline and calcified cartilage may occur in this location and produce considerable restriction of joint motion. Exostoses about the knee joint spring mainly from the mesial and lateral surfaces. Spur formation from the upper margin of the mesial condyle is very common, producing a cylindrical mass of bone which may extend upward in the substance of the abductor longus muscle for a distance of 1 to 4 in. Tumors springing from the lateral surface are usually hemispherical in shape but may be attached by either a narrow or broad pedicle. Fracture is not uncommon in case of a narrow pedicle, leaving a movable mass of bone in the soft parts. Retrogressive changes usually develop in such detached masses, leading to gradual reduction in size. Extensive irregular tumor formation is very common in connection with the upper end of the shaft of the tibia. Occasionally short spurs form which are directed downwards. Because of their exposed position such tumors are subject to traumatism and may give rise to considerable discomfort (Fig. 34).

Marked irregular growth of the upper end of the fibula is common. Its epiphysis is not affected. The enlargement may extend downward 2 or 3 in. below the epiphyseal line (Fig. 34). Irregular nodular tumor formation is also common at the mesial side of the lower end of the tibia, producing deformity and frequently much discomfort as a result of irritation by the shoes. Tumors arising from the lateral surface of the tibia may produce erosion and curvature of the lower metaphysis of the fibula, which is itself infrequently the seat of such growths. As maturity is reached and ossification of the cartilaginous cap occurs, synostosis of the bones is the usual result. The shortened lower end of the fibula leads frequently to considerable angulation of the plane of the ankle joint with eversion of the foot (Fig. 27) and lateral shifting of the astragalus so that it articulates with the under surface of the external malleolus.

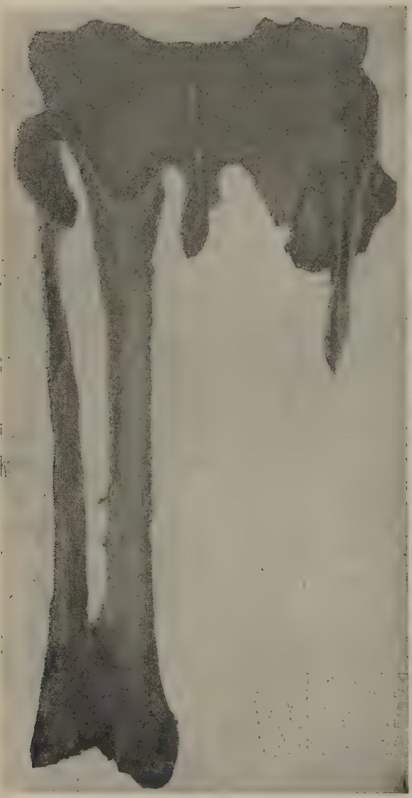

Fig. 34.—Tibia and fibula in male, age forty, with multiple cartilaginous exostoses. Fusion of lower ends and obliquity of ankle joint due to short fibula.

The bones of the feet are less frequently involved than those of the hands, but may be the seat of either enchondromas or exostoses, which are frequently small in size.

Roentgen-ray Findings.—The Roentgen-ray appearance of multiple cartilaginous exostoses is characteristic. The end of the shaft from which the exostosis springs is usually expanded and its thinned cortex directly continuous with that of the tumor, the outline of which is sharply circumscribed. Usually there is a narrow cortical shadow about the periphery of the tumor and the interior casts a faint, spongy shadow. Where a thick zone of calcification is present about the limits of the tumor, a dense, sometimes stippled, shadow may be present (Fig. 29). There is no evidence of bone erosion, which is of great service in differentiating between exostosis and sarcoma. If the roentgenogram is taken in the axis of the exostosis, so that the shadows of the tumor and the shaft overlap, an area of marked reduction in density, corresponding to the outline of the base of the tumor, will be seen. This is due to the fact that the bone in the exostosis casts a fainter shadow than would have been cast by normal cortex which it has replaced. There is no evidence of new bone formation in the interior of the shaft, but frequently its cancellous trabeculæ may be atypically arranged or absent in rather extensive areas. It is extremely rare to see extension of the exostosis onto the epiphysis, although the epiphyseal lines may be obliquely displaced from arrested growth on the side of the exostosis.

Complications.—Complications arising from exostoses are comparatively rare. Malignant degeneration is so rare that it scarcely deserves consideration. Chiari observed sarcoma arising from the upper end of the humerus in a case of multiple cartilaginous exostoses with marked involvement of practically all the long bones of the body. Sarcoma or chondrosarcoma occasionally arises from enchondromas which are associated with multiple cartilaginous exostoses.

Complications arising from pressure upon neighboring structures are of the most importance. Cervical, branchial or lumbosacral neuritis may be produced by tumors arising from the lateral portions of the spine (Fig. 32). Rarely intraspinal tumors develop which compress the spinal cord. Ochsner and Rothstein described an exostosis which sprang from a vertebral arch in the cervical region and produced root and cord symptoms. Bevan operated on a similar case, where the tumor was located in the lower thoracic region. Intestinal obstruction, which at first is chronic, but may become acute, may arise from large tumors of the pelvis. The writer saw a fatal termination from this cause in a case that had shown obstructive symptoms for several years. Interference with pregnancy and labor is common in large pelvic exostoses. Erosion of the popliteal artery by tumors in that location may lead to aneurism formation. Von Statzer has collected 16 such cases. Paralysis of the perineus nerve may result from large exostoses of the upper end of the fibula. Tumor formation in connection with the bones of the feet may interfere greatly with walking.

Diagnosis.—A well-marked case of multiple cartilaginous exostosis is scarcely to be confused with any other condition. Its onset during childhood with the gradual development of multiple painless tumors from the ends of the shafts and the associated disproportioned shortening of certain bones are characteristic. Rarely a large tumor early in the course of the disease, when other changes are slight, may be confused with periosteal sarcoma, but the absence of pain, the slowness of growth and absence of evidences of cortical erosion and radiating ossification in the tumor should make a differentiation comparatively easy.

Treatment.—Nothing can be done to arrest the course of the disease. Since growth usually ceases when adult life is reached and retrogression may even occur in some of the tumors, a conservative policy should be pursued from the surgical standpoint. Indications for removal of the tumors are for cosmetic reasons, because of the complications that arise, and because of the pain that they may occasion. Large tumors about the knee and shoulder most frequently necessitate operative removal. Fingers and toes may occasionally have to be amputated when the seat of large deforming growths.

A tumor may be removed at any time, regardless of age, with small chance of recurrence if the operation is properly performed. The growth should be freely exposed and detached at its insertion, taking great pains to include and go outside of the cartilaginous covering. If all of the cartilaginous covering is removed, recurrence will not take place.

Ulnar flexion of the hand and curvature of the radius may be remedied by cuneiform osteotomy of the lower end of the radial shaft. Occasionally this should be supplemented by osteotomy of the curved ulna. Osteotomy for genu valgum may be necessary. In case of massive tumor formation of the upper end of the fibula, complete excision of the affected region may be indicated.

Extensive resection of the shaft to which exostoses are inserted should rarely be practised. This has been done repeatedly under the mistaken diagnosis of sarcoma.

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Multiple Enchondromas

This is a disturbance of enchondral ossification which results in retardation of longitudinal growth and the formation of enchondromas in the ends of the shafts of bones. It is allied to multiple cartilaginous exostoses, in that the disturbance is of the cartilaginous epiphyseal line, but the distribution of the lesions and the character and location of the tumors are quite different in the two conditions.

Etiology.—The condition is hereditary, but not to the same extent as are multiple cartilaginous exostoses. Where a family tendency is marked, enchondromas and exostoses are likely to occur together. There are no other factors which have a bearing upon its development. The disease is congenital. The lesions are sometimes recognized at birth, especially when they are confined to one or two of the extremities.

Clinical Manifestations.—There are two fairly distinct forms; one in which multiple enchondromas develop in the ends of the shafts, especially of the short bones of the hands and feet, and the other in which the long bones of one or two of the extremities are involved, to which Ollier has given the name of dyschondroplasia. In the first form of multiple enchondromas the lesions usually first appear between the ages of four and eight years, and lead to the formation of oval expansions or nodular tumors, principally of the proximal ends of the phalanges or the distal ends of the metacarpals and metatarsals (Fig. 35). Meyerding reported a case of extensive multiple enchondromas beginning in adult life. Occasionally the end of a long bone of the extremities is the seat of similar enlargement, in which event it may be shorter than normal and somewhat curved. The ribs are the most frequently affected of the trunk bones, especially at their costochondral junctions. The tumors steadily enlarge during the period of growth of the skeleton, some of them very slowly, others more rapidly, so that masses varying in size from a pea to a child's head may be present by the time that adult life is reached. Contrary to the findings in exostoses, growth continues in many of the enchondromas after the age of maturity. Longitudinal

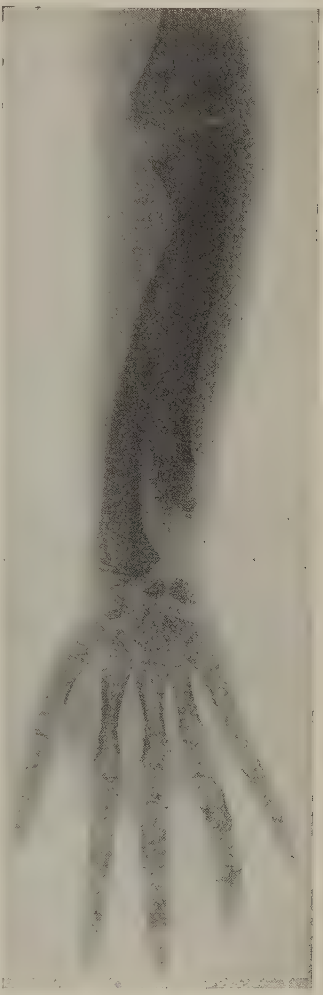

FIG. 35.—Enchondromas of lower end of ulna and of finger bones.

growth may be interfered with and shortening of the extremity result.

Pathology.—There is a failure of substitution of cartilage of the epiphyseal line by bone, so that islands are carried into the growing ends of the shaft. Here cartilaginous growth continues with resultant tumor formation. Expansion of the end of the shaft results, but the tumor usually bulges more on one side and may completely erode the cortex, in which event the cartilaginous mass projects into the soft parts with only a perichondral fibrous covering. Hyaline cartilage makes up the bulk of the tumor. It usually extends to the epiphyseal line, but does not involve the bony epiphysis, except by displacement when the tumor is of large size. Scattered areas of calcification and ossification may occasionally develop within its substance. When large, softening and liquefaction with cyst formation may occur. An enchondroma may produce spindle-shaped enlargement of a short bone of the hand, new bone forming from the periosteum as the tumor erodes the compacta from within. Occasionally the enchondromas are situated about the periphery of the shaft, eroding the bone from within outward as they grow, and forming nodular projections about the surface. In extreme cases they may be pedunculated, resembling cartilaginous exostoses.

A section of the tumor shows its hyaline cartilaginous nature, and any ossification or calcification which may be present is irregularly distributed, especially along the course of connective tissue septa and blood-vessels which run through its interior. Enchondromas situated about the ends of the long bones may produce curvature or angulation of the extremities, such as genu valgum, flat foot or luxation of the radius. Enchondromas are usually firm in consistency, but vary from soft, semi-fluctuating masses, where mucoid degenerated cartilage is present, to stony hard masses where extensive calcification has occurred. The color varies greatly in the secondary changes; where degeneration has not occurred, the tumor has a bluish, opalescent appearance; soft, degenerated portions may have a fibrous or gelatinous appearance, and the calcified parts are yellowish-white in color. Where the entire shaft of the bone is expanded, there may be a bony shell about the face of the tumor even when it has reached considerable size.

Complications.—Malignant degeneration of enchondromas is not uncommon. Weber and Huber found softening of an enchondroma with metastases in the lymph glands and internal organs. Microscopically the enchondromas are indistinguishable from benign cartilaginous lesions, as mitotic figures and heavy staining nuclei do not occur. Myxomatous degeneration of an enchondroma of the femur was observed by Frangenheim, necessitating amputation. Nehrkorn and Kryger reported sarcomatous degeneration of enchondromas. Weber saw a malignant enchondroma and exostoses combined.

Diagnosis.—The diagnosis is based upon the multiplicity of the lesions, beginning in childhood, the preference for the short bones, especially of the hands, the slow growth and marked variation in size, and the Roentgen-ray appearance of the lesion. Roentgenograms show oval or somewhat lobulated circumscribed areas of reduced density in the interior of the end of the shaft. Where the tumor projects markedly from the periphery, cortical shadows may be entirely lacking. In case of calcification of the interior, irregular, dense, scattered areas of increased density are to be seen. The individual lesions, when small and interiorly situated, are to be distinguished with difficulty from cysts, fibromas and giant cell tumors or the bone.

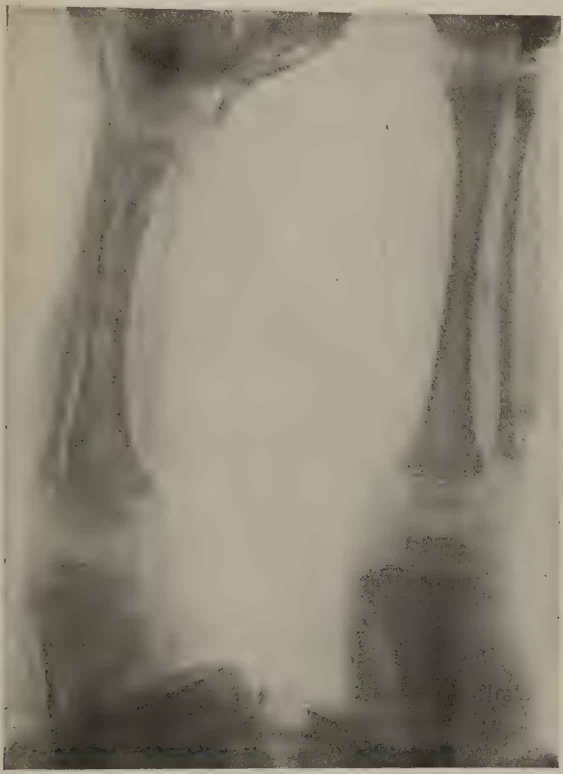

Fig. 36.—Dyschondroplasia in two and one-half year old child, affecting ends of left femur and tibia and external supracondylar region of right femur.

Treatment.—Operative removal is frequently indicated because of deformity and interference with function. Large enchondromas of the fingers and toes may necessitate amputation. Small tumors may be treated conservatively by excision of projecting masses and scraping out the interior of the bone, which is followed by restoration of the shaft. Occasionally complete excision of a segment may be indicated with replacement by a bone transplant. Rapid increase in size of an enchondroma should arouse suspicion of malignancy and call for surgical interference.

Dyschondroplasia.—Dyschondroplasia is a term applied by Ollier to the second form of enchondromatous growth disturbance. In it the long bones of one, or at most two, of the extremities show marked disturbance of longitudinal growth with slight expansion and enchondroma formation in the metaphyses. The distribution of the lesions in the extremities is irregular. Lower extremities are most frequently involved, but it may affect only upper extremities. One leg, an arm and a leg on the same side or on opposite sides, and both legs with unequal involvement are the commonest distributions. The short bones of the hands and feet are usually free. The lesion is of the cartilaginous epiphyseal line and both epiphysis and end of the shaft may be affected. The involved extremity is usually short at birth, but when in the leg it frequently remains unnoticed until the child begins to walk. As growth continues, the shortening and consequent limp become more marked and slight curvature of the affected bones may develop. By the fifteenth or eighteenth year a difference of 3 to 6 in. may exist in the length of the extremities, especially the lower, in which case marked tilting of pelvis and scoliosis result. Growth of the enchondromatous ends of the shafts usually ceases when adult life is reached. Usually a slight expansion of the ends of some of the shafts can be observed, but tumor formation of appreciable size is extremely rare.

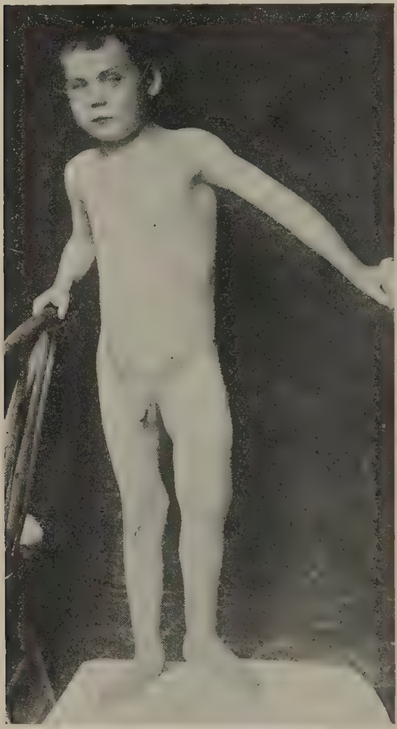

Fig. 37.—Dyschondroplasia of left lower extremity, age seven years.

Pathology.—The end of the shaft of the growing bone is slightly expanded for a distance of 1 to 3 in. from the epiphysis, depending upon the bone affected, and frequently more on one side than another. Section of the involved region shows a thin layer of cortical bone and a large mass of hyaline cartilage filling the interior. The epiphyseal line may be straight or slightly curved and irregular, as the result of more marked involvement in some portions than in others. A similar but much less marked involvement may be found in the epiphyses, where small islands of cartilage may extend into the center of ossification from the epiphyseal line. Irregular small islands of calcification or ossification may be scattered throughout these masses of hyaline cartilage which fill the interior of the ends of the shafts. In some cases the cartilaginous inclusions may affect only one side of the metaphysis, the remainder consisting of normal cancellous bone. The lesion appears to be a congenital disturbance on the part of the epiphyseal line, in which normal metamorphosis of cartilage preparing the way for ossification at the end of the shaft does not occur; consequently replacement of the growing cartilage does not take place, which leads to accumulation of cartilage in the metaphysis. The scattered areas of calcified cartilage probably represent islands of the zone of calcification which are carried into the depths by the surrounding hyaline cartilage. Ossification may occur in these calcified centers with increasing age. Molin and Wittek have reported on the condition in detail, and the author has studied two similar cases. Figure 36 is a roentgenogram of the extremities of a two and one-half year old boy whose left leg was 2 in. shorter than the right. The ends of the left femur and tibia show irregular reduction in density with longitudinal streaking of the ends of the shafts of femur and tibia, and the lateral portion of the end of the shaft of the right femur shows similar changes. The upper extremities were uninvolved. Figure 37 shows a seven year old boy whose left leg had been short since birth and measured 3 in. shorter than the right. The ends of the shafts of the left femur, tibia and fibula were the only bones affected, the foot being entirely free. Figure 38 shows the roentgenologic appearance at the knee. Scattered dense areas can be

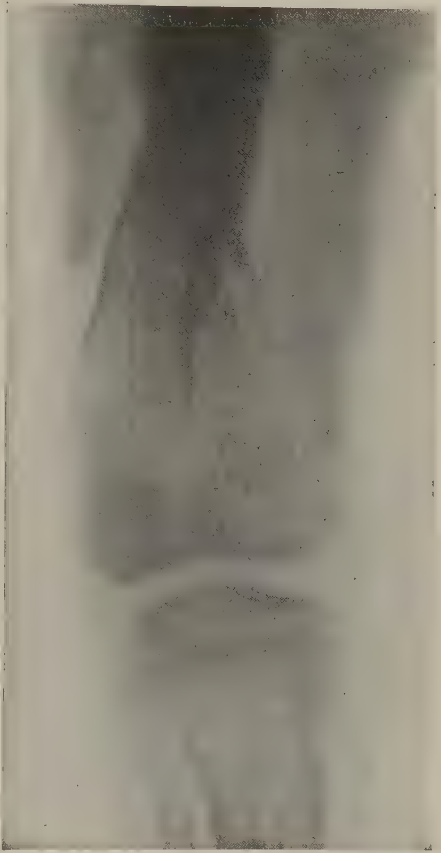

Fig. 38.—Roentgenogram of knee region of case in Fig. 36.

seen through the greatly rarefied ends of the shafts and epiphyseal involvement with dense flecks is seen in the external condyle and lower epiphysis of the tibia and fibula. A section excised from the length of the involved area of the lower end of the tibial shaft showed the interior of the bone filled with hyaline cartilage containing a few scattered areas of calcification and ossification. One one-hundredth of a grain of phosphorus twice daily for nine months caused the appearance of a dense line from ossification in the juxta-epiphyseal portions of the shafts and enlargement of the shadows cast by the irregularly distributed islands of calcification and ossification (Figs. 39, 40).

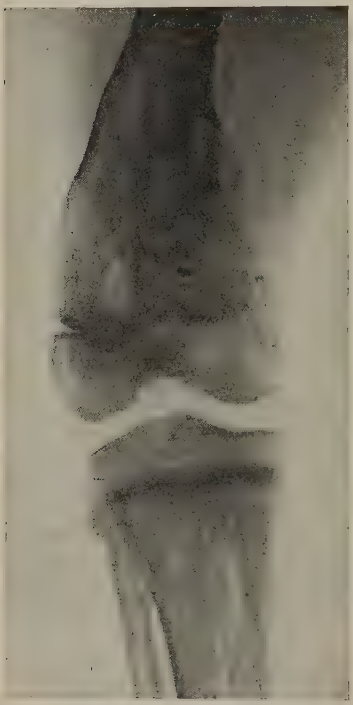

Fig. 39.—Same as Fig. 38 after nine months' treatment with phosphorus.

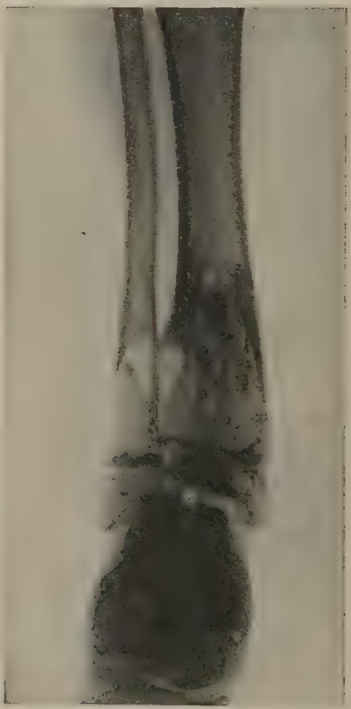

Fig. 40.—Lower ends of leg bones after nine months' treatment with phosphorus.

Diagnosis.—The multiplicity and distribution of the lesions make the diagnosis comparatively easy. It has to be distinguished especially from fibro-cystic disease of the bone. The latter condition rarely affects more than one metaphysis and does not produce shortening. It usually extends farther into the shaft and produces greater thinning of cortex away from the epiphyseal line. The epiphysis is never affected in bone cyst, as it may be in dyschondroplasia.

Treatment.—About the only treatment is that directed toward correction of the inequality in length when the involvement is of the lower extremity. The epiphyseal line of the lower end of the femur on the unaffected side may be obliterated by operation at the proper age to bring the two extremities out of equal length when adult life is reached. Osteotomy is occasionally indicated for the correction of deformities. There is no tendency for fractures to occur through the involved metaphyses, and phosphorus administered over a prolonged period did not seem to accelerate the longitudinal growth of the leg in the case (Figs. 39, 40) where it was used.

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CHAPTER CIX

THE SURGERY OF TENDONS

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Anatomy and Physiology of Tendons.—To deal successfully with tendons the surgeon must know their anatomy and physiology. By their anatomy I refer not merely to the course and insertion of the tendons, their relationship to fascial planes, and their action, but to the finer details of their structure, and the exact mechanism of their motion. The tendon far from being a simple structure, is exceedingly complex and is one of the body tissues most susceptible to trauma. The tendon consists of bundles of fibrils all running in the longitudinal direction, surrounded by a connective tissue envelope which penetrates between the bundles dividing the tendon into smaller subdivisions. The tendon cells have elongated nuclei whose axes correspond to the line of traction to which the tendon is subjected. Where the tendon turns a corner and a certain amount of friction results, these tendon cells change their character; the nuclei instead of being long become round and closely resemble those of cartilage cells. The transition of these cells to real cartilage and to the development of a sesamoid bone follows as a natural sequence of events. The cells on the surface of the tendon differ somewhat from those in the deeper portion and seem to be particularly adapted to the gliding motion. Whenever they are injured either by rough handling or by drying, an adhesion is almost certain to result. In operative work on tendons, therefore, the gliding surface of the tendon must be carefully protected from all trauma except at the point where adhesion of the tendon either to another tendon or to the bone is the goal of the operation, and then the gliding cells must be removed by thorough scraping. It is quite essential in handling the tendon that the fibrils be held together and not be torn apart. The tendon should therefore never be touched with the ordinary mouse-tooth forceps, since they disrupt the tendon fibrils; the gloved finger moistened with saline or a rounded clamp which holds the fibrils together should be used instead.

The function of the tendon is the transmission of the contractile force of the muscle and its gliding mechanism is therefore of the utmost importance. The tissue most concerned in the gliding mechanism is the loose, fatty mesh work, rich in elastic fibers which surrounds the tendon on all sides. Owing to its elasticity it enables the tendon to glide to and fro beneath the rigid fascia. To this tissue the term *paratenon* has been applied. The tendon sheath is a closed sack containing fluid interposed between the tendon and the fascia or the bone at any place where the tendon changes its direction. The sheath serves as a fluid buffer to diminish friction at this point. Where the tendon runs a straight course no sheath is present. The term tendon sheath is frequently incorrectly applied. The surgeon often speaks of the sheath of the Achilles tendon for instance, although no true sheath is present. The sheath properly conceived corresponds to a joint in that it is a sharply circumscribed cavity containing a synovial-like fluid. It does not give the tendon an increased range of motion since the tendon glides as freely above the sheath as within it.

When the sheath of a tendon is opened and the tendon lifted out, a delicate connective-tissue membrane is seen connecting the tendon with the floor of the sheath. This structure, known as the *mesotenon*, transmits blood-vessels to the tendon and corresponds roughly to the mesentery of the intestine(Fig. 41). That portion of the tendon into which it is inserted is termed the hilus. It is always on the surface of the tendon least exposed to friction. Here the connective tissue of the mesotenon expands on the surface of the tendon forming the *epitenon*, and sends connective-tissue strands between the tendon bundles, thus forming the *endotenon* (Fig. 42). When stretched to the maximum, the mesotenon of the extensor proprius hallucis tendon is 3 to 4 cm. wide. The mesotenon is continuous with the connective tissue surrounding the tendon above and below the sheath. When the tendon is allowed to drop back into place the mesotenon adapts itself to its narrow quarters by forming numerous folds which cover the floor of the sheath. The mesotenon differs radically in one respect from the mesentery; it may be absent. Thus the tendon of the tibialis posticus is always without a mesotenon: the flexor longus hallucis tendon in 70 per cent. of the specimens examined, the flexor longus digitorum tendon in 50 per cent. When the mesotenon is absent, it is represented at each end of the sheath

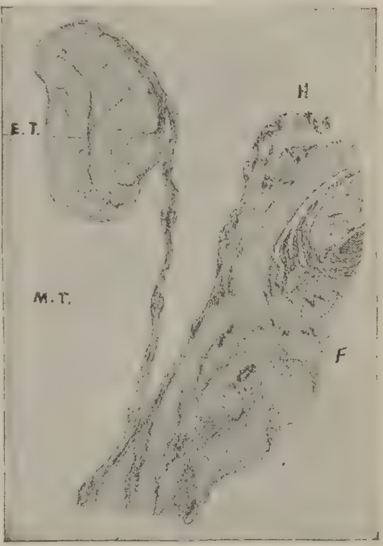

Fig. 41.—Microscopical cross section of the tendon of the tibialis anticus (eight months infant) 0.5 cm. above the intermalleolar line. Leitz obj. 1, oc. 0, tube 140. Previous to fixation, the sheath had been slit open, the tendon lifted out so as to stretch the mesotenon, and was held in this position during fixation. The section demonstrates the relation between the tendon hilus, mesotenon, and floor of the sheath. *E.T.* Endotenon; *M.T.* mesotenon; *F.* floor of the sheath; *H.* hilus of the tendon.

by a short membrane which, depending upon its shape, may be termed a vinculum triangulare or quadrangulare. Usually in addition to these vincula there are one or more fine strands connecting the tendon with the sheath, vincula filiformia. These residual mesotena are usually coupled to one another on the surface of the tendon corresponding to the hilus by a thin band of connective tissue (epitenon) in which the blood-vessels ramify.

At the upper and lower poles of the tendon sheath there is a special mechanism termed the *plica*. This structure acts as a kind of valve which permits the free motion of the tendon and at the same time preserves the tendon sheath intact. The plica is a downward projection of the paratenon within the sheath. It may be either a simple fold (plica simplex) corresponding to the deflection of the conjunctiva from the lid to the eye-bulb, or it may be a double fold (plica duplicata) which looks like a thin veil and divides the upper pole of the sheath into two pockets (Fig. 43). The significance of the plica is first understood when the motion of the tendon is studied. This has been done by observations during operations, by animal experiments, and by cadaver tests. They show that during the contraction of a muscle and the consequent gliding of the tendon, the plica follows the motion of the tendon either by an elongation of

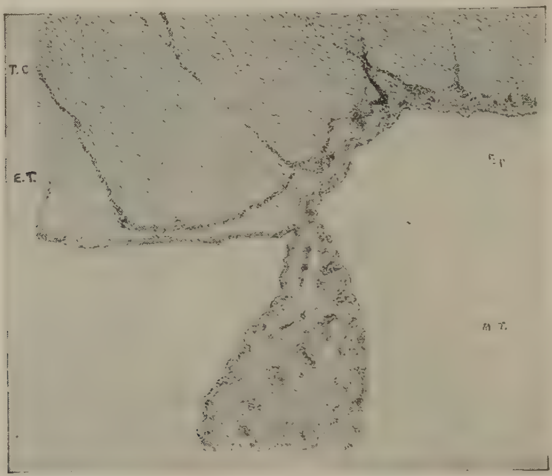

FIG. 42.—Microscopical cross section through the hilus of the extensor longus hallucis tendon (adult). Leitz obj. 3, oc. 1, tube 140. *T.C.* Tendon cells; *E.T.* endotenon, *Ep.* epitenon; *M.T.* mesotenon.

The mesotenon expands at its insertion into the tendon, forming a delicate tightly adherent connective-tissue enveloping layer—the epitenon. From the epitenon, connective-tissue septa extend into the tendon, separating it into larger and smaller bundles. These septa I have termed the endotenon (old terminology: peritenonium internum), as opposed to the esotenon, the connective tissue coating the surface of the tendon. Within the sheath the esotenon is represented by the epitenon and the mesotenon; above the sheath and at the two portals of the sheath by the paratenon.

its folds (plica simplex) or by a kind of invagination (plica duplicata). An elongation of 4 or 5 cm. may occur without rupture of the delicate lining of the sheath.

In former studies of tendons, they have frequently been characterized as avascular structures. This is however, quite incorrect. The tendons possess an extensive system of blood-vessels which always run on that surface of the tendon least exposed to friction. The vessels here from an anastomotic net and send numerous branches into the connective tissue between the tendon fibrils. The main blood-vessel of the tendon usually runs through the lowermost fibers of the corresponding muscle. Other vessels of the tendon course through the mesotenon and the paratenon. A few smaller vessels pass into the tendon at the point of insertion. The tendons are most vascular

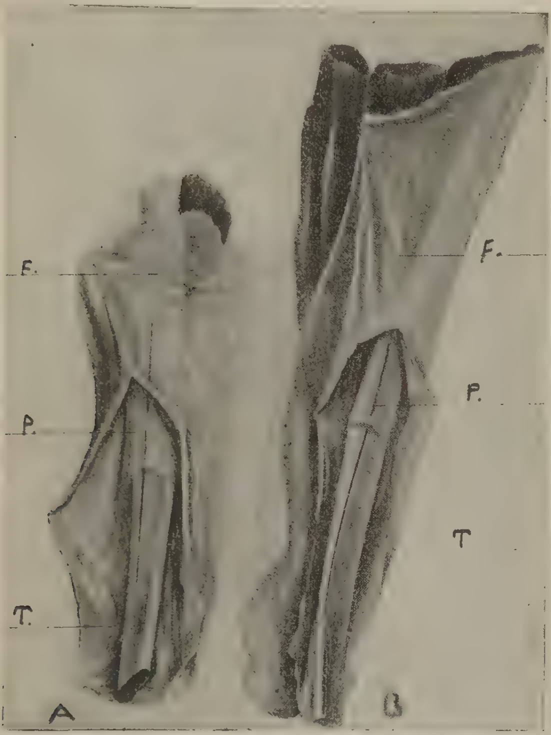

Fig. 43.—Two drawings of the tendon of the tibialis anticus: (*A*) During the phase of muscular relaxation (the foot in equinus). (*B*) During the phase of muscular contraction (the foot in calcaneus). *F*. Fascia; *P*. plica; *T*. tendon free in sheath. In each instance the superficial pocket of the sheath has been opened up to the upper pole, a bristle has been passed into the deep pocket until it encounters the resistance of the reflection of the plica to the tendon (that is the upper pole of the deep pocket).

In *A* the deep pocket is 1.0 cm. deep; in *B*, 2.8 cm.

This type of gliding mechanism is found frequently in young individuals and in most adults.

near the insertion of the mesotenon; that is, near the hilus. Here the numerous branches of the mesotenon (Figs. 44, 45) ramify in the epitenon and send numerous fine twigs into the tendon. An accurate knowledge, not merely of these general principles, but of the vascular system of each individual tendon is of practical value in tendon operations. The tendon of the flexor longus hallucis, for instance, derives its blood supply regularly from a vessel which, reaching the tendon via the

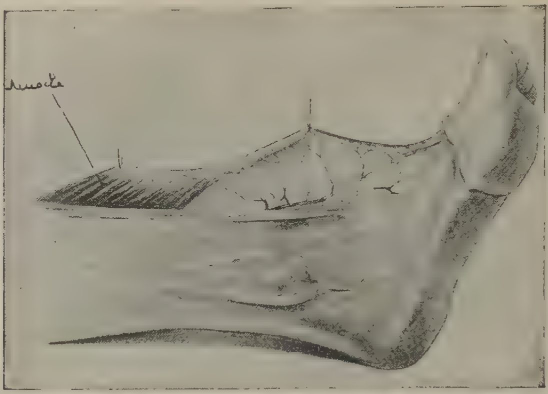

Fig. 44.—The blood-vessels of the extensor proprius hallucis, injected with gelatin-cinnabar. The sheath has been opened and the tendon lifted out so as to put the mesotenon on the stretch. From the dorsalis pedis artery five branches run through the mesotenon to the hilus of the tendon, and form an extensive anastomosis in the epitenon. (The number of branches varies in different individuals, but the principle of the vascular system is constant.)

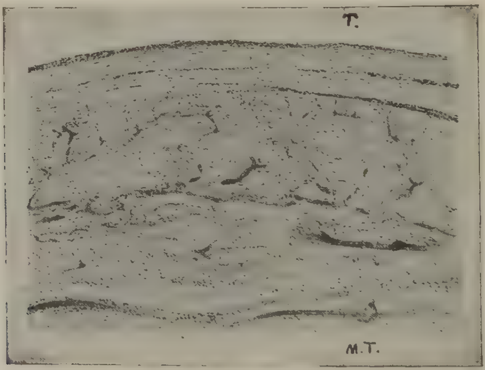

Fig. 45.—Longitudinal microscopical section through the tendon of the extensor proprius hallucis (six months infant). The vessels were injected previous to fixation with a suspension of Turnbull's blue in glycerin. The drawing shows the vessels in two successive serial sections, each 50 microns thick. Note the numerous vessels passing from the mesotenon (*M.T.*) into the tendon. Near the friction surface (*T*) of the tendon, however, no vessels are present.

proximal vinculum or the lowermost fibers of the muscle, runs downward on its posterior surface and anastomoses with a corresponding vessel of the distal vinculum. In transplanting the tendon to replace a paralyzed Achilles tendon, the surgeon can, with a little care, avoid injuring the dorsal vessel and thus insure the viability of the tendon.

The tension of a tendon is of as great practical significance as the mechanism of its motion. It is obvious that when a tendon is transplanted or sutured, the operator should attempt to restore the normal tension, yet, despite this self-evident fact, no attempt was made to investigate the physiology of tendon tension until 1913. Then it was shown that though the tension varies directly with the size and strength of the muscle, one important measurement remains constant, namely, when under anesthesia with the muscles completely relaxed, the extremity is held in such position that the origin of the muscle and the insertion of the tendon are brought as near together as possible, then the tension of the tendon is zero. For instance, if the foot is held inverted and dorsiflexed, if in other words, the origin and insertion of the tibialis anticus muscle are brought as near together as possible, then the tension of the tibialis anticus tendon is zero. This fact is of easy application to all tendon operations, since, to restore normal tension the operator need only hold the limb in the position indicated and suture the tendon with just enough force to render its course a straight one. If the normal tension is not restored, two mistakes may be made; either the tendon may be sutured with too little tension and thus a certain amount of slack is created which the muscle will find it hard to take up, or, it may be sutured with excess tension which strains the muscle fibers and causes their degeneration.

Diseases of Tendon and the Tendon Sheath.—Probably the most frequent disease affecting the tendons is an inflammation of the tendon sheath, known as *tendovaginitis crepitans* or *tendovaginitis sicca.* It is characterized by the deposition of fibrin within the sheath. This causes a peculiar crepitation, audible and palpable when the tendon moves to and fro. The inflammation may extend beyond the sheath into the paratenon. In fact, despite the name tendovaginitis which would indicate a disease of the sheath, it often develops where no sheath is present, as in the case of the Achilles tendon. The condition is caused by over-exertion and it subsides when the tendons are kept at rest.

A much rarer type of tendovaginitis crepitans occurs in a chronic or recurring form. In one case which came under the author's observation, the disease had been present on and off for 12 years. Its onset followed shortly after a severe tonsillitis and the disease was eradicated by removal of the infected tonsils. An acute serous synovitis also occurs. The tendon sheath is distended by fluid and feels like a small sausage. This type of synovitis is seen in children as a rheumatic manifestation, and occasionally in adults as a complication of gonorrhea.

Acute purulent inflammation of the tendon sheaths of the fingers is a common occurrence and unless treated with painstaking care, usually leads to the formation of dense adhesions or to the destruction of the tendon. Frequently an involvement of the tendon sheath can be prevented by early accurate diagnosis and incision of the purulent focus. When the sheath is involved, numerous small incisions should be made along the lateral aspects of the sheath. The anterior mid line incision so commonly used, is always to be avoided.

Tuberculosis of the tendon sheath occurs in two forms: The serous type, known as compound ganglion, characterized by a stubborn serous effusion and the formation of rice bodies, second, the fungating or caseating form. The serous type may be cured by puncture and the injection of iodoform in oil combined with general hygienic treatment and the administration of tuberculin. The second type always requires complete excision for its cure. The functional results are as a rule, good. Syphilis may also produce a chronic inflammation of the tendon sheath, but its occurrence is rare.

Tumors may arise from the connective tissue or from the synovial membrane of the sheath. The former are usually sarcomata. Of the synovial tissue tumors, about 120 have been reported. They have been assigned a number of different names: Granuloma, myeloma, xanthoma, etc. Tumors are treated by local extirpation. The prognosis is usually favorable.

Mention must also be made of small connective tissue tumors of the tendon sheath which give rise to the phenomenon of *snapping finger.* In this condition the flexion and extension of the finger meets at one point with a resistance which, when overcome by the contraction of the muscles, results in a sudden snap of the finger. The snapping may also be caused by a thickening of the tendon itself or general thickening of the membranes. The treatment usually resolves itself into the operative removal of the obstruction to the motion of the tendon. Occasionally immobilization will effect a cure.

Ganglion.—This name is given to certain cystic bodies which develop in the neighborhood of tendons. Their mode of origin is doubtful. They form hard, smooth, rounded tumors of varying size. Usually they are found on the dorsum of the wrist. They contain a clear gelatinous fluid.

Some ganglions may be obliterated by continuous pressure or by a sharp blow. Occasionally puncture of the cyst with scarification of its interior will make it disappear. Usually however, the sack must be completely excised before the patient is cured.

Injuries to Tendons.—Strains.—A tendon may be injured by a sudden over-exertion or by excessive use. What the pathological changes are, can only be surmised, since biopsies have not been performed on cases of this type. Clinically they evidence themselves by pain on use of the injured tendon, by tenderness on pressure, and discomfort when the tendon is subjected to passive stretching. They are treated by (1) immobilization in a position which relaxes the tendon, (2) by the firm pressure of adhesive plaster strapping, (3) by heat, best applied by a flat-iron.

Dislocation.—A tendon may be displaced from its normal groove by a sudden muscle contracture when the limb is in an unusual position. The tendon is usually replaced with ease and held in position with adhesive plaster. If the dislocation becomes habitual, the groove for the tendon should be deepened by an appropriate bone plastic and the restraining ligaments reinforced by a fascial transplant.

Subcutaneous Rupture.—A sudden force which stretches a tendon beyond its physiological length may rupture the tendon or tear it away from its insertion together with a small fragment of bone (tear fracture). The tendons most frequently injured are the supraspinatus, the extensors of the fingers at the distal interphalangeal joint, and the plantaris tendon. Nonoperative treatment is usually successful. The part is splinted in such a way as to relax the injured tendon and approximate the torn edges. Thus, for a tear fracture of the supraspinatus, the arm is splinted in maximum abduction, for a rupture of the extensor of the finger, the finger is kept hyper-extended, for a tear of the plantaris, the foot is kept in equinus and the knee flexed. In addition local pressure is applied by adhesive plaster to prevent an exudate from forming. If the tendon fails to unite, open operation is indicated (see traumatic suture of tendons).

Wounds of Tendons.—These occur in peace as well as in war. In industrial surgery they constitute an important problem since total loss of function ensues if the tendon is not properly repaired. The tendons of the forearm, hand and fingers are most frequently injured, since they are most exposed to accident.

Surgical Procedures.—**Traumatic Suture of Tendons.**—When tendons have been divided, the operator must decide first of all when to unite them. Whenever the wound is due to some clean, sharp object, such as a knife or a pane of glass, tendon suture should be performed as soon as possible after the accident. If however, the wound is large, badly contaminated and there is extensive destruction of tendon tissue requiring a tendon plasty, then operation should be postponed until all signs of infection have disappeared. Between these two extremes, lie many cases in which the decision is bound to be difficult. In general, however, it is preferable to err on the side of early operation.

When a tendon has been divided the proximal end invariably retracts owing to the contraction of the muscle, leaving a gap of varying size between it and the distal stump. Immediately after division, this gap may be only ½ in. or 1 in., but within a few weeks it is likely to be 2 or 3 in. when we are dealing with the long tendons of the fingers and even more when dealing with the long tendons of the foot. To find the retracted tendon at the time of operation, several methods may be attempted; the muscle may be milked downward in the direction of the tendon or an Esmarch bandage may be applied beginning near the proximal attachment of the muscle and forcing it downward at each turn. Felizet suggests in cases of division of one of the flexors of the fingers, strong hyperextension of the neighboring fingers. All these methods however fail in many cases and then there is nothing to do but prolong the incision in the direction of the retracted tendon until it has been exposed. The plan of burrowing for the tendon with some instrument is unwise, since it does damage not only to the tendon, but also to the surrounding tissues.

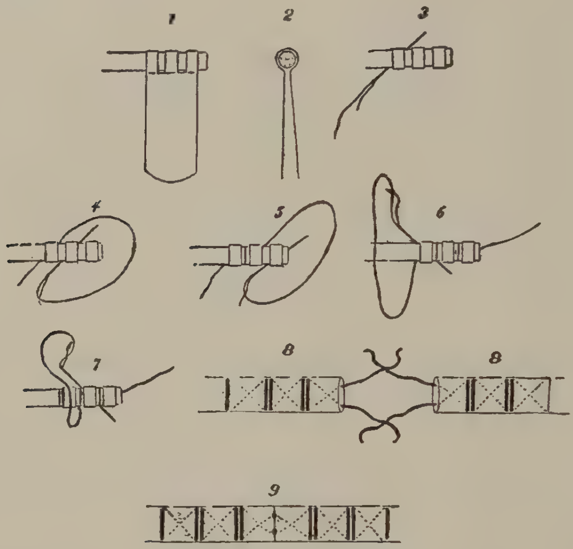

FIG. 46.—Diagram illustrating the insertion of the Bunnell stitch. (American Jour. of Surgery.) 1. The clamp applied, viewed from the side. 2. View of the free end of the tendon with the clamp applied. 3. Starting the stitch with the first needle. 4. Second stitch with the first needle. 5. Third stitch with the first needle. The needle emerges at the cut edge of the tendon. 6. Starting the first stitch with the second needle. 7. Second stitch with the second needle. 8. The stitches have been inserted into both tendon ends preparatory to knotting them. 9. The sutures have been knotted and the ends cut off short, bringing the tendons into accurate apposition.

In performing a tendon suture, certain general principles must be carefully observed which are applicable to all operations on tendons. The tendon must always be regarded as an exceedingly friable, delicate tissue which must never be exposed to the air more than a short time. It should be carefully protected from all other forms of traumatism. Under no circumstances should the tendon be grasped with a mouse-tooth forceps except at that point where an adhesion is desired. During the time that the tendon is exposed, it should be frequently bathed with normal saline to prevent the gliding cells on its surface from drying.

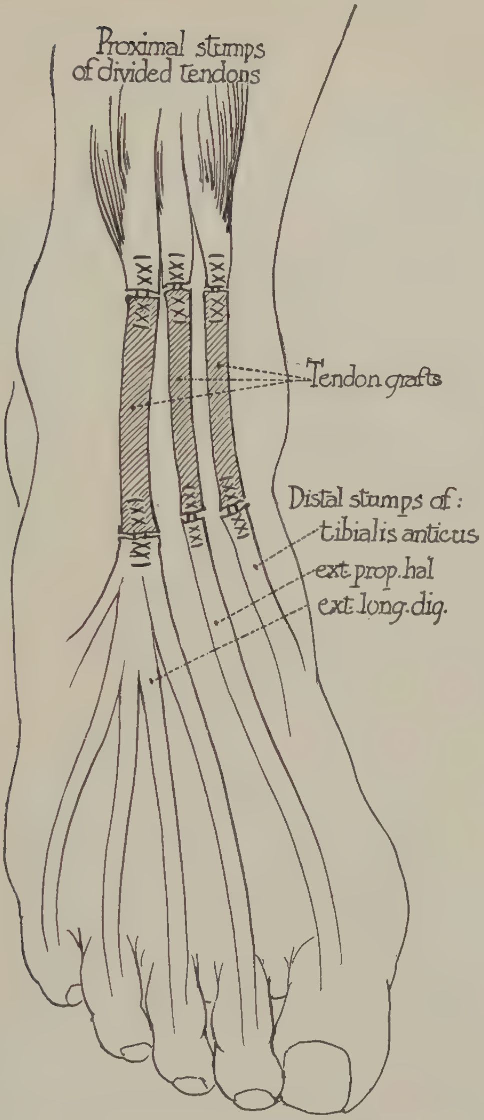

Fig. 47.—Diagram illustrating the implantation of tendon grafts taken from the peroneus longus muscle to bridge the gap between the divided tendons on the dorsum of the foot. Each graft about 3 inches long was sutured to the tendon stumps by means of fine chromic gut. The channel for the reception of the grafts corresponded accurately to the normal situation of the tendon sheaths.

The tendon suture must have sufficient mechanical security to hold the two tendon ends in firm apposition. A great variety of sutures have been described but probably the best is that of Bunnell. Two silk sutures, each about 10 in. long are used, one for each stump. Each is threaded with two straight needles. The stitch is begun about ⅜ in. from the cut surface. Several crisscross stitches are taken through the thickness of the tendon (Fig. 46), the free ends are brought out at the cut surface. The ends of the sutures are then knotted so that the knots lie between the tendon stumps. For the more exact execution of this suture, Bunnell devised an ingenious clamp (Fig. 46). It is however, possible with a little practice, to insert the suture without the device. After suture of the tendons, the operator should always attempt to restore the gliding mechanism of the tendon. This can frequently be done by turning a pedunculated flap of fascia so as to cover over the line of suture, or if the sheath has been injured, reuniting the edges by very fine stitches. Catgut may be used for this purpose but silk should be used for the tendon suture itself. The silk suture will frequently be extruded unless the wound is quite aseptic but the union of the tendon will ensue before this has occurred.

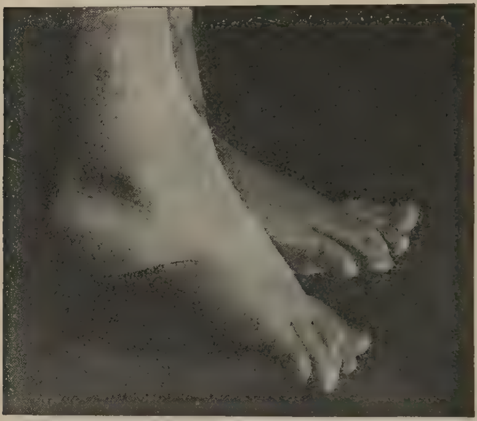

Fig. 48.—Double photograph (two exposures on one plate) illustrating the range of active motion at the ankle subsequent to the insertion of free tendon transplants to bridge the gap between the divided dorsal flexors of the foot. The tendons function with almost the normal strength and the patient walks without a limp. Operation February, 1920; photographed November, 1920.

The Free Transplantation of Tendons.—When tendon suture is to be postponed either because of extensive sepsis or destruction of tendon, or in those cases in which the primary suture has not held, a subsequent operation should be undertaken to repair the damage. In the past various forms of tendon lengthening have been proposed; these consisted in turning down flaps out of the tendon in such a way as to bridge the gap separating the tendon stumps. Glück used strands of catgut to bridge the gap between the tendons, Lange favored silk. A more physiological procedure is the free transplantation of tendon to replace the missing portion. The graft should be taken preferably from the same individual and the tendon should be wherever possible, of the same diameter as the one to be replaced. When working on the hand, the palmaris longus is usually the best for transplantation purposes. A portion of the flexor carpi radialis may be split off by following one of the natural cleavage lines of the tendon. In the foot one of the tendons of the extensor brevis digitorum or the peroneus longus or a portion of the Achilles tendon may be used to advantage. The operation consists in the exposure of the two tendon stumps through appropriate incisions. These should always be planned in such a way as not to lie directly over the tendon. When working on the fingers, the incision should always be lateral, never in the mid-line. After the stumps have been exposed, freed from their adhesions and the ends freshened, the same stitch is inserted which has already been described for immediate tendon suture. The distance between the tendon stumps is then measured off. A corresponding length of the tendon to be transplanted is threaded at each end with the usual stitch and is then drawn downward along the old course of the tendon which has been destroyed. Whenever possible this course should correspond accurately to the sheath of the tendon. The passage is best constructed by burrowing with a small dressing forceps or artery clamp. It should be sufficiently wide to accommodate the transplanted tendon

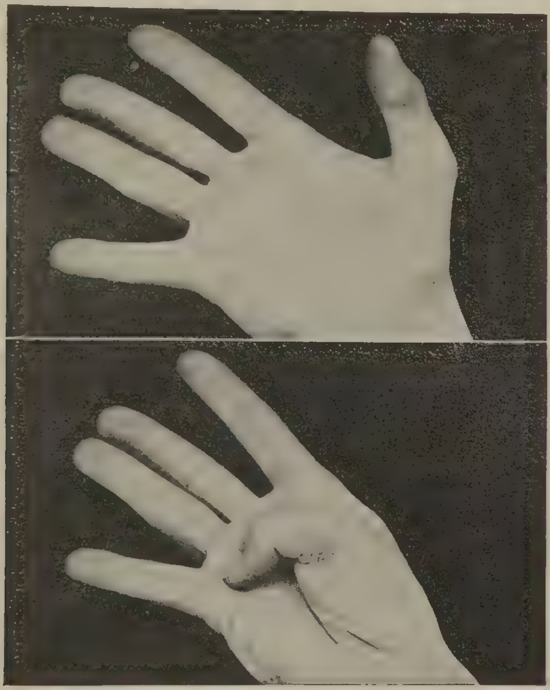

FIG. 49, *A* and *B*.—These photographs illustrate the range of active motion of the thumb a year and one-half subsequent to the implantation of a tendon graft bridging the gap between the divided ends of the flexor longus pollicis tendon. Injured October 7, 1918, operated upon December 13, 1918, photographed June, 1920.

without pinching it at any point. When the new tendon has been brought into its reconstructed channel, the sutures are tied (Fig. 47). Exercises should be begun two weeks after the implantation. Figures 48, 49 illustrate the results of free transplants in the case of the long flexor tendon of the thumb and the extensor tendons of the foot.

Tendolysis.—Sometimes when a tendon has been injured the tendon ends unite without open operation but they become densely adherent to the bone or to the sheath at the point of injury. After a tendon suture, dense adhesions may develop which prevent the tendon from functioning. In other instances, the tendons particularly at the wrist, become matted together by scar tissue. In all these cases a tendolysis is indicated, that is, an operation which frees the tendon from adhesions. Whenever possible, this operation should be performed under local anesthetic, since the active contraction of the patient's muscle is a

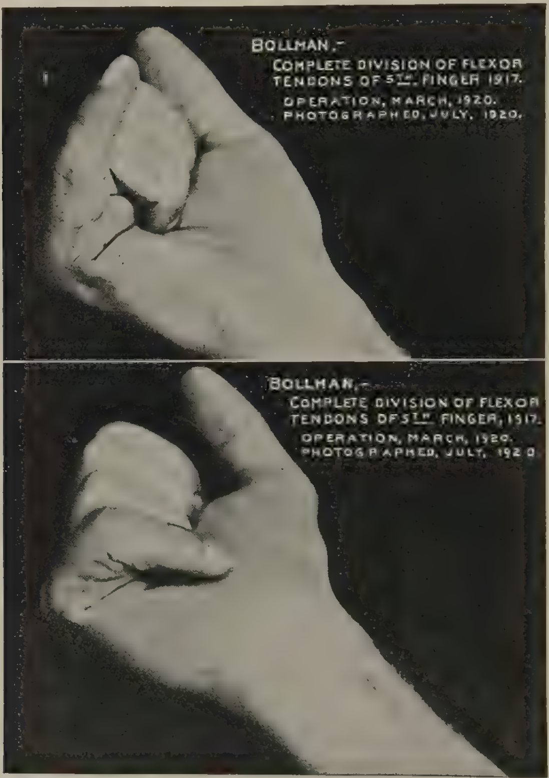
BOLLMAN.-
COMPLETE DIVISION OF FLEXOR
TENDONS OF 5TH. FINGER 1917.
OPERATION, MARCH, 1920.
PHOTOGRAPHED, JULY, 1920.
BOLLMAN.-
COMPLETE DIVISION OF FLEXOR
TENDONS OF 5TH. FINGER, 1917.
OPERATION, MARCH, 1920.
PHOTOGRAPHED, JULY, 1920

FIG. 50, *A* and *B*.—These two photographs illustrate the range of motion of the fifth finger four months subsequent to tendolysis. The tendons of the fifth finger were freed from the bone to which they were adherent and surrounded by a fascial transplant whose gliding surface was turned outward to prevent the formation of adhesions subsequent to the operation.

valuable factor in breaking down the adhesions. The incision should be so placed as not to lie over the site of the injury. In freeing the tendon, the most accurate anatomical knowledge and painstaking care are necessary since many of the normal landmarks are obscured by scar tissue. As soon as the main adhesions have been divided, the patient is requested to contract the muscle in question and this frequently breaks through the smaller adhesions. If the area of adhesion is small, further operative measures are usually unnecessary. The patient need only exercise the tendon assiduously for a number of weeks immediately subsequent to the operation. If however, the adhesion was very extensive, it is advisable to transplant a piece of fascia so placed as to have its smooth gliding surface turned outward. The fascia is made to surround the scarred portion of the tendon and serves to reconstruct the gliding surface of the tendon. The fascial edges are united by a running suture and at each end it is firmly united to the tendon. The fascia is supposed therefore to move with the tendon. After this procedure as in the case of a simple tendolysis, post-operative exercises should begin on the day subsequent to the operation (Fig. 50).

Tenotomy and Tendon Lengthening.—The purpose of a tenotomy is a lengthening of the tendon by the simplest possible method. Its rationale depends upon the fact that two tendon ends which do not separate more than about 1 in. will unite within a few weeks if the surrounding tissues are preserved intact. The new tissue—the tendon callus—is derived partly from the tendon itself, partly from the proliferation of the surrounding connective tissue, particularly the paratenon.

The operation should be performed with a delicate tenotome and should be an accurate division of the tendon with a minimal cutting of the surrounding tissues. Whether the operator cuts with the blade towards or away from him, is a matter of individual preference. It is however, of great importance that in dressing the wound, the gap between the tendon ends be left free of pressure. If the operator in his zeal for accurate hemostasis, places a little pressure pad between the tendon ends, he will effectually prevent the tendon from reforming.

Tenotomy should never be performed in cases of spastic paralysis since spastic muscles contract unduly and draw the tendon ends so far apart that union is impossible. A number of methods have been devised to overcome this difficulty. Sir Robert Jones takes two strong stitches through the tendon about ½ in. apart, cuts between them and knots the suture to prevent retraction beyond the desired distance. Another method long in use is the step-like division of the tendon, a longitudinal splitting of the tendon with two short transverse cuts at each end which divide opposite halves of the tendon. After the tendon has been lengthened the desired amount, the two overlapping halves should be united by a few interrupted stitches. The double L-shaped incision of Hibbs also gives good results.

Tenotomy or tendon lengthening is indicated when a tendon has become so short as to interfere with function of the limb. The short Achilles tendon of the typical hemiplegic, is a conspicuous example. Much critical thought should be given to each case of short tendon since occasionally, despite the imperfect function the short tendon is more effective than a normal one would be. For instance, if a paralyzed leg is shorter than the normal, a slight grade of equinus improves the gait and prevents a tilting of the pelvis.

Subsequent to the operation, the limb should be immobilized in plaster of Paris or an appropriate splint for a period of three weeks. Active exercises to develop the strength of the tendon should be begun after this period.

Tendon Transfers.—These operations are particularly applicable to the treatment of the residual paralyses of poliomyelitis and of extensive gunshot injuries. The purpose of the operation is to shift the attachment of the tendon in such a way as to transform the action of the muscle itself. A tendon which originally acted as an evertor of the foot, for instance, the peroneus longus, can be transformed into an invertor by shifting it to the inner side of the foot, or a muscle which previously bent the knee or flexed the wrist can, by altering its insertion, be changed into an extensor. A most striking physiological fact is the ease in which the brain can accommodate itself to these anatomical changes. In children particularly it is astonishing with what readiness the function of the muscle can be completely changed within a short period of time. In these operations as well as in tendon suture and free tendon transplants, there is the utmost need for "atraumatic" technique. In addition the operation must meet the following demands:

1. It must whenever possible restore the normal relationship between the tendon and the sheath.
2. The course of the tendon from its original site to that of the paralyzed tendon must run through tissue adapted to the gliding of the tendon. Injury to the periosteum or the boring of a hole through fascia or interroseous membrane is inconsistent with this demand.
3. The normal insertion of the tendon must be imitated wherever possible by implanting the tendon directly into bone or cartilage, preferably at the insertion of the paralyzed tendon.
4. The normal tension of the transplanted tendon must be reestablished and the physiological length of the transplanted muscle thus maintained.
5. The line of traction of the transplanted tendon must be such as to enable it effectively to do the work of the paralyzed tendon.

These general principles have developed as the result of the clinical and experimental studies of the last 30 years. The first surgeon to attempt a tendon plastic was Nicoladoni, who, in 1880 attempted to replace a paralyzed Achilles tendon by means of the peroneal tendons. Unfortunately, owing to the discrepancy between the strength of the implanted tendon and that of the paralyzed, the result was poor and for 16 years little was heard of tendon plastics. Then Drobnick suggested the advisability of implanting the transferred tendon into the bone instead of into the paralyzed tendon. His suggestion was eagerly seized upon by Lange of Munich who developed a system of tendon plastics in which the transplanted tendon was lengthened by the addition of a suitable number of heavy silk sutures. In this way a periosteal or bone implantation was rendered possible in every instance. In contra-distinction to this system of Lange, Vulpius insisted on the advisability of sewing tendon to tendon. For almost 15 years the surgery of tendon plastics consisted in a vigorous controversy between these two factions. Neither party to the controversy was able to present convincing experimental proof of the correctness of his views. In 1913 the first significant research appeared from Lange's clinic. A serious attempt was made to determine the cause of post-operative adhesions to tendons and it was found that these could always be prevented by preserving the normal relationship between the tendon and the sheath. This research confirmed the suggestion of Biesalski, who, in 1910 had advised withdrawing the paralyzed tendon from its sheath and drawing the transplanted tendon downward through the vacant sheath. The Biesalski method was developed and modified by the author and resulted in a series of tendon operations in which each step of the operation was correlated with the known facts of tendon anatomy and physiology.

Although a large number of operations are possible experience has shown the following to be of the greatest value:

1. Transfer of the peroneus longus to replace the paralyzed tibialis anticus (paralytic flat foot).
2. Transfer of the tibialis anticus to replace the evertors of the foot (paralytic club foot).
3. Transfer of the peroneus longus or brevis combined with that of the flexor longus hallucis to replace the paralyzed Achilles tendon (paralytic cavus)
4. Transfer of the ham-string muscles to replace the paralyzed extensors of the knee.
5. Transfer of the flexors of the wrist to replace the paralyzed extensors.
6. Transfer of the flexor sublimis tendon to replace the divided flexor tendons of an adjacent finger.
7. Transfer of the extensor communis digitorum tendon of the index finger to replace the divided extensor tendon of the thumb, ring or little finger.

1. *Paralytic Flat Foot.*—The operation usually indicated is the transfer of the peroneus longus to replace the paralyzed tibialis anticus. The chief anatomical difficulty in the execution of the operation is the fact that the peroneus longus lies in a fascial compartment separated throughout its extent from that of the tibialis anticus.

To overcome this difficulty, a fascial plastic is necessary. The first step of the operation is the preparation of the implantation site for the peroneal tendon. An incision about 2 in. long, bowed with the convexity toward the sole of the foot, is made over the insertion of the tibialis anticus. The paralyzed tendon is exposed at its insertion, slit longitudinally for about 1 in., and the subjacent bone or cartilage of the internal cuneiform is grooved with a small gouge for the reception of the peroneal tendon. It is advisable to prepare the implantation site as the first step of the operation so as to avoid exposing the peroneal tendon to the air more than is absolutely necessary. The method heretofore commonly practiced of freeing the peroneal tendon and wrapping it up in gauze while the implantation site is being prepared, is to be condemned as unphysiological, since the gliding cells on the surface of the tendon are readily traumatized by contact with any foreign substance. It is quite essential for the successful gliding of the transplanted tendon, that it be transferred from its original site to the new one within a very few minutes and with minimal handling. The cuneiform bone is purposely traumatized so as to encourage its osteogenetic activity. Adhesion between the tendon and the traumatized bone occurs in less than 16 days and the union by that time is sufficiently firm to permit active motion of the transplanted tendon.

The second incision, about 1 in. long, is made near the upper pole of the tibialis sheath, about 2 in. above the tip of the internal malleolus. The sheath is opened just enough to permit the passage of a probe downward through the sheath and out at its lower extremity (Fig. 51). The tip of the probe is then seen to emerge just at the insertion of the tibialis anticus. The probe should be bent slightly near the tip and should be passed in such a way as to lie on the anterior surface of the tendon. Unless this precaution is taken, the probe has a tendency to slip off to the side and to traumatize the mesotenon or vascular membrane which connects the tendon with the floor of this sheath. Into the eye of the probe, is threaded a strong silk or linen suture. The two free ends of the suture are passed through the eye so that the loop can be utilized at a later step of the operation to draw the tendon downward without the necessity of knotting. Even a small knot is bound to traumatize very much more than the smooth loop. The probe is drawn entirely through the sheath leaving the guide suture in place.

The third incision must be a long one, since the peroneal tendon must be exposed up to the middle of the calf if it is to be given the proper line of traction. A common mistake is to free the tendon only a short distance above the external malleolus thus rendering its course transverse rather than vertical. Its action under such circumstances is just the opposite of the required; it everts the foot instead of inverting it. The incision runs from the middle of the calf downward to the external malleolus, around it, and down to the cuboid bone. It is made slightly S-shaped, the upper part of the incision nearer to the tibia, the lower part near the fibula. This is done so as to facilitate the execution of the fascial plastic. The skin and subjacent tissues are retracted from the underlying fascia until not only the peroneal muscles but also the muscles of the anterior fascial compartment are visible. A longitudinal slit is made in the fascia over the anterior compartment and over the lateral compartment (Fig. 25). Depending upon the age of the patient, these openings in the fascia should be from 2 to 4 in. long. With a little practice it is easy to estimate their length correctly. At each end of the longitudinal slit, small transverse incisions are made to permit the eversion of the fascia (Fig. 53). The object of this procedure is to evert the deep layer of the fascia which is coated with paratenon or gliding tissue, and over this gliding tissue, the peroneal tendon can be made to pass from one fascial compartment into the other. If this were not done, adhesions would almost certainly occur, since traumatism to the fascial septum, as shown both experimentally and in secondary operations, gives rise to adhesions which would impede the gliding of the tendon. The two fascial edges are united by means of a fine silk suture inserted in much the same way as the serosa stitch of gastro-enterostomy. It is very important that the surface of the fascia coated with gliding tissue be entirely untouched by any instrument or gauze. Only the edge which is inverted by the stitch is grasped by the forceps. The suture itself when properly inserted, does not

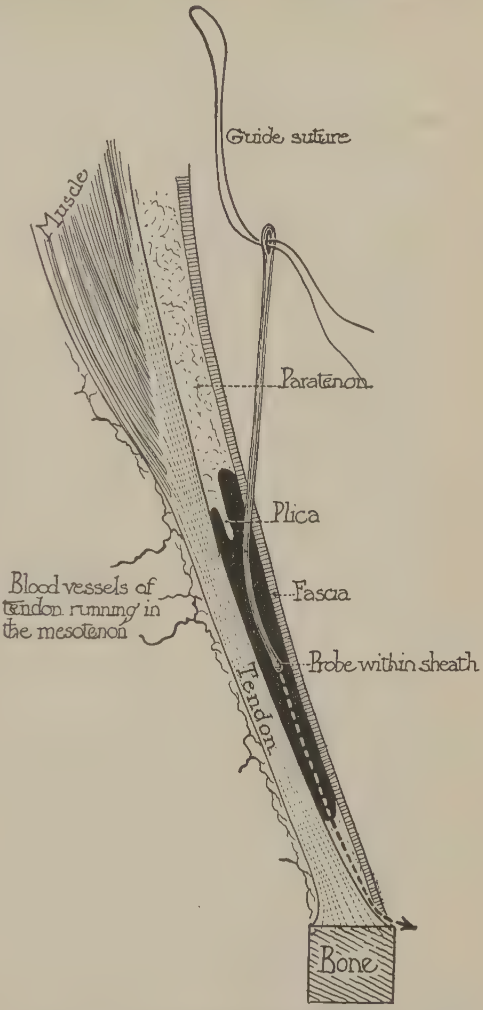

Fig. 51.—Diagrammatic longitudinal section through the tibialis anticus tendon illustrating the manner of passing the probe downward through the tendon sheath. The dotted line and the arrow indicate the passage of the probe through the lower pole of the tendon sheath and out near the insertion of the tendon.

show at all. I have found the Cushing modification of the Lembert suture more practical than any other. Instead of tying the suture at the lower end of the fascial flap, it is advisable to take an additional stitch so as to tie at a somewhat lower point and thus avoid any possible contact of the tendon with the knot. When the suture is being inserted, the assistant prevents drying of the tissues by frequent applications of warm physiological saline solution.

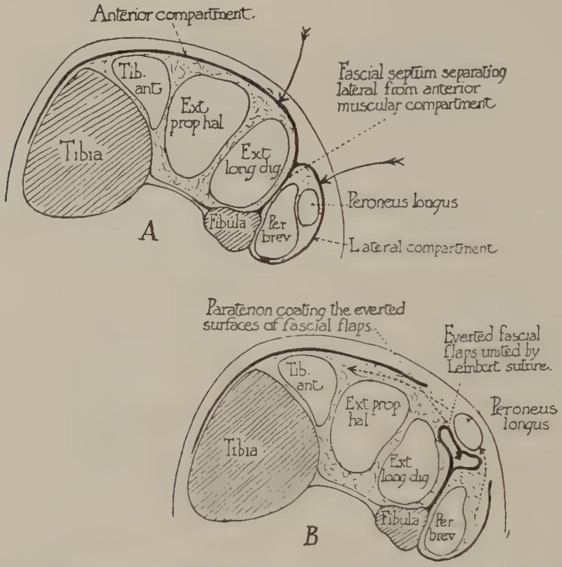

FIG. 52.—Diagrammatic cross section of the calf illustrating the division into the anterior and lateral muscular compartments by a fascial septum (*A*). To transfer the peroneus longus tendon from the lateral compartment to the anterior, the fascia is incised at the points indicated by the arrows and the fascial edges are then everted and brought together by a suture as indicated in *B*. The paratenon or gliding tissue which coats the deep surface of the fascia prevents the formation of adhesions.

The eye probe must now be passed from the upper pole of the tibialis sheath beneath the fascia and out in the region of the fascial plastic. The upper end of the guide suture lying in the tibialis sheath, is drawn beneath the fascia by means of the probe. The guide suture thus runs from the fascial plastic beneath the fascia curis into the tibialis anticus sheath, downward through the sheath and out near the insertion of the tibialis tendon. It is everywhere in contact with gliding tissue and by drawing the peroneal tendon along this course, it will make its pathway correspond to the normal. Only after all these preparatory steps, is the operator ready to expose the peroneal tendon. The tendon is freed by prolonging the fascial incision already made over its upper end, downward until the sheath

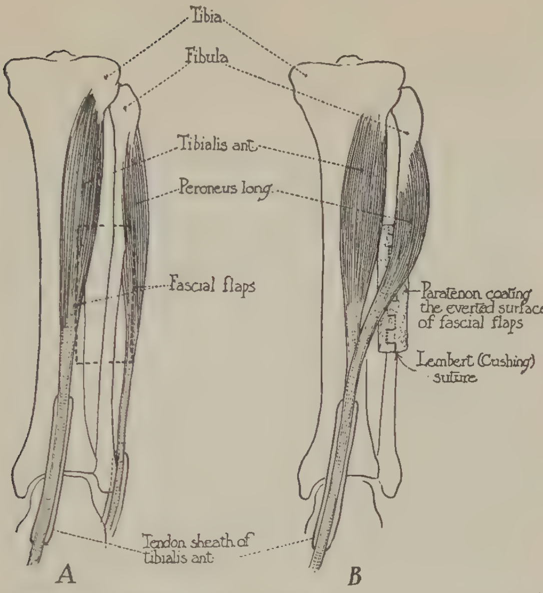
Tibia
Fibula
Tibialis ant.
Peroneus long.
Fascial flaps
Paratenon coating the everted surface of fascial flaps
Lembert (Cushing) suture
Tendon sheath of tibialis ant.
A
B

FIG. 53.—Diagrams illustrating the transfer of the peroneus longus to replace the tibialis anticus. *A*, Shows the form and location of the fascial flaps which are everted to facilitate the passage of the peroneus longus from the lateral muscular compartment to the anterior. *B*, Illustrates the course of the peroneal tendon over the fascial flaps after they have been everted and united by a Lembert suture. The paratenon coating the everted deep layer of the fascia prevents the formation of adhesions.

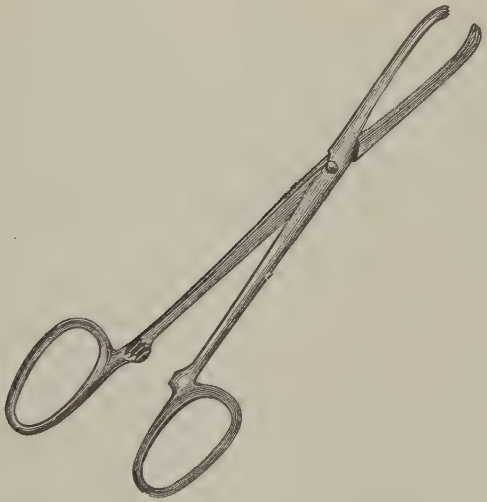

FIG. 54.—The author's tendon clamp.

has been opened and then along the sheath to the groove in the cuboid bone where the peroneal tendon passes into the sole of the foot. It is at this point that the tendon should be divided, since experience has shown that it is then just long enough to reach to its new point of insertion. The tip of the divided tendon is grasped with the tendon clamp (Fig. 54) and a No. 2 chromic suture is inserted as indicated in Fig. 55. The ends of the suture are left long to facilitate drawing the tendon downward through the sheath of the tibialis anticus. The end of the tendon is trimmed carefully to make it smooth and rounded so as not to traumatize the tibialis sheath in its passage. As rapidly as possible the tendon is now freed up to the lowermost muscle fibers and drawn over the fascial bridge gently downward through the tibialis sheath (Fig. 53). While doing this the first assistant must hold the tendon in the air by means of any smooth instrument so as to prevent the tendon from buckling. The operator must grasp the end of the tendon with a forceps and start it on its pathway, since otherwise torsion is likely to occur. This step of the operation, more than any other, requires practice and team play for its rapid, gentle execution. No force should be used and the act of transplanting should not require more than five seconds.

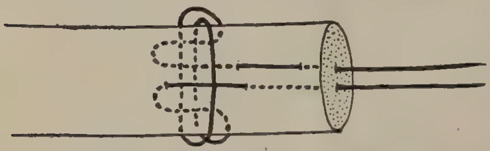

Fig. 55.—The fixation suture. This is inserted by using a suture with a straight needle at each end. With the first needle, two transverse stitches are taken through the tendon and then a longitudinal which is brought out at the cut surface of the tendon. With the second needle a longitudinal stitch only is taken which emerges on the cut surface. This fixation suture will stand great tension without tearing.

The long incision is closed at once to prevent further exposure of the tendon to the air, then the second incision is closed. The operator is then ready to attach the tendon to its new insertion. The ends of the chromic gut suture are passed into a stout cervix needle with which several firm bites are taken through fascia and cartilage. The fixation should be such as to draw the tendon directly against the bed in the internal cuneiform bone already prepared for it. The two-halves of the split tibialis tendon are united to the tendon so as to give additional security. This method of fixation meets the demands of mechanical security and also of physiological fixation, since the traumatized bone cells react to hold the tendon secure. To determine the degree of tension under which the tendon should be sutured, the operator has recourse to the rule already referred to, namely, that when under anesthesia the origin and insertion of a muscle and its tendon are approximated, the tension of the tendon is zero. The foot is therefore inverted and dorsiflexed and the peroneal tendon is pulled upon with just enough force to render its course straight. It will be found that its length, if it has been divided at the cuboid groove, is just sufficient to make it reach the bony implantation site prepared for it.

The operation is concluded by suturing the first skin incision. The foot is then immobilized in plaster of Paris in a position of inversion and dorsiflexion (Figs. 56–58).

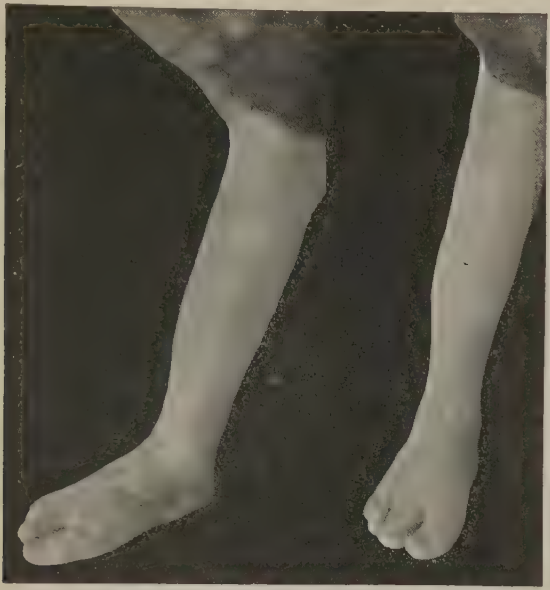

Fig. 56.—(at left). Paralytic flat foot due to paralysis of the tibialis anticus and posticus.

Fig. 57.—Same patient after transfer of the peroneus longus to replace the paralyzed tibialis anticus. The planovalgus deformity has been entirely corrected.

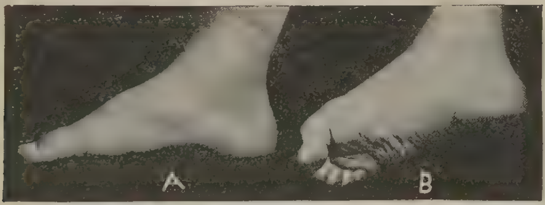

Fig. 58.—*A*, the effect of transplantation of the peroneus longus through the sheath of the tibialis anticus in a case of severe paralytic flat foot due to paralysis of both tibialis anticus and tibialis posticus. Note the complete absence of planus deformity. Operation June, 1918; photographed July, 1920.

B, Same patient as in *A*, illustrating the degree of voluntary inversion subsequent to the transplantation of the peroneus longus through the sheath of the tibialis anticus.

2. *Paralytic Club Foot.*—Transplantation of the tibialis anticus is done to replace the evertors of the foot. A horseshoe-shaped incision is made over the insertion of the peroneus tertius, the skin flap being directed upward. The tendon of the peroneus tertius is split at its

insertion and a groove cut in the fourth and fifth metatarsal bones for the reception of the tibialis tendon.

The second incision is made along the course of the tibialis anticus tendon from a point slightly proximal to the upper pole of the sheath downward to its insertion. The fascia is incised just above the sheath and is retracted toward the outer side of the leg so as to expose the extensor proprius hallucis and the extensor longus digitorum (Fig. 59). An eye probe threaded with a guide suture is now passed downward along the course of the extensor longus digitorum tendons and made to enter its sheath (Fig. 60). This is a simple procedure if the operator is careful to make the tip of the probe hug the tendon. This is best done by making a small hole in the paratenon which surrounds the tendons into which the tip of the probe is inserted. On entering

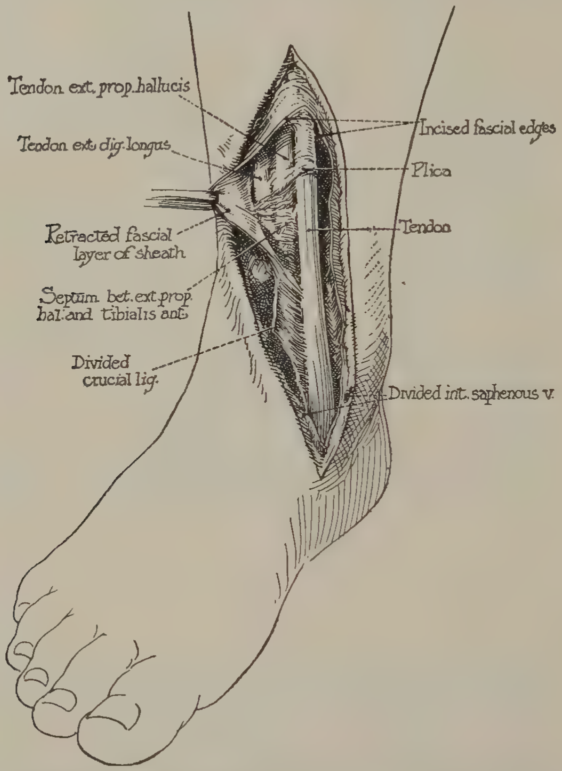
Tendon ext. prop. hallucis
Tendon ext. dig. longus
Retracted fascial layer of sheath
Septum bet. ext. prop. hal. and tibialis ant.
Divided crucial lig.
Incised fascial edges
Plica
Tendon
Divided int. saphenous v.

Fig. 59.—Transfer of the tibialis anticus to replace the paralyzed evertors of the foot in a case of paralytic club-foot. The drawing illustrates the second incision which exposes the tibialis anticus tendon. The fascia is retracted by a clamp exposing the extensor longus digitorum tendons above their sheath. The probe inserted at this point passes downward along the tendons into the sheath of the extensor longus digitorum and out near the insertion of the peroneus tertius. (From Mayer, Orthopedic Treatment of Gunshot Injuries.)

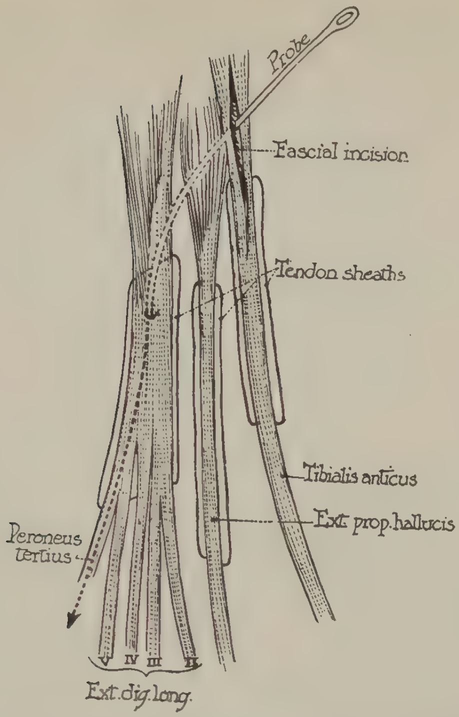

Fig. 60.—Diagram illustrating the passage of the probe through the sheath of the extensor longus digitorum for the transference of the tibialis anticus to the outer side of the foot. The dotted line and the arrow indicate the direction of the probe through the sheath and its emergence at the insertion of the peroneus tertius.

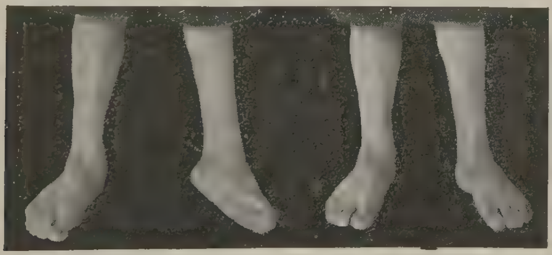

Fig. 61.—(at left). Paralytic club-foot before treatment.

(At right). Same patient one year after transplanting the tibialis anticus through the sheath of the extensor longus digitorum to replace the paralyzed evertors of the foot. The transplanted tendon functions with sufficient strength to maintain normal muscle balance.

the sheath, the probe finds its way readily downward and is made to emerge over the peroneus tertius tendon which runs through the same sheath as the long extensor (Fig. 60). The probe is pulled through bringing the guide suture after it. The tibialis anticus sheath is split open its entire length and the tendon cut away at its point of insertion. Unless the full length of the tendon be secured, it will prove too short to reach to the outer side of the foot. It is advisable to take a small bit of cartilage or bone with the tendon since this renders the union at the new point of implantation, particularly firm. The tendon is threaded with the usual fixation suture, freed from its mesotenon and by means of the guide suture, drawn downward through the sheath of the extensor longus digitorum and made to emerge at the insertion of the peroneus tertius. Here it is fastened to the groove already made in the bone. The operation is concluded by suturing fascia and skin. The leg is immoblized with the foot in maximum eversion and dorsiflexion. (Fig. 61).

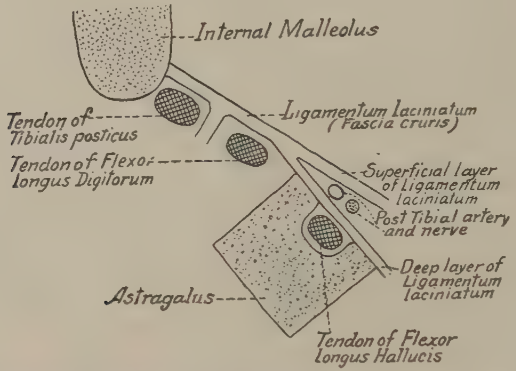
Internal Malleolus
Ligamentum laciniatum (Fascia cruris)
Tendon of Tibialis posticus
Tendon of Flexor longus Digitorum
Superficial layer of Ligamentum laciniatum
Post Tibial artery and nerve
Deep layer of Ligamentum laciniatum
Astragalus
Tendon of Flexor longus Hallucis

Fig. 62.—Diagram illustrating the fascial planes covering the tendon of the flexor longus hallucis at the ankle. To expose the tendon the superficial layer of the deep fascia (ligamentum laciniatum) must be incised, the posterior tibial artery and nerve drawn toward the ankle and then the deep layer of the fascia opened before the tendon is exposed to view. (From Mayer, Orthopedic Treatment of Gunshot Injuries.)

In mild cases of club foot, the extensor proprius hallucis may be used instead of the tibialis anticus. The operation is performed in exactly the same manner.

3. *Paralytic Cavus* (*Paralysis of the Gastrocnemius and Soleus*).—Transplantation of the peroneus longus or brevis combined with that of flexor longus hallucis is done to replace the paralyzed Achilles tendon. This operation is indicated in cases of mild cavus deformity. When the cavus deformity is more marked, bone operations are necessary.

After a preliminary division of the plantar fascia, of the plantar muscles, and if necessary of the long plantar ligaments, the foot is wrenched thoroughly so as to restore the normal form. Two trans-

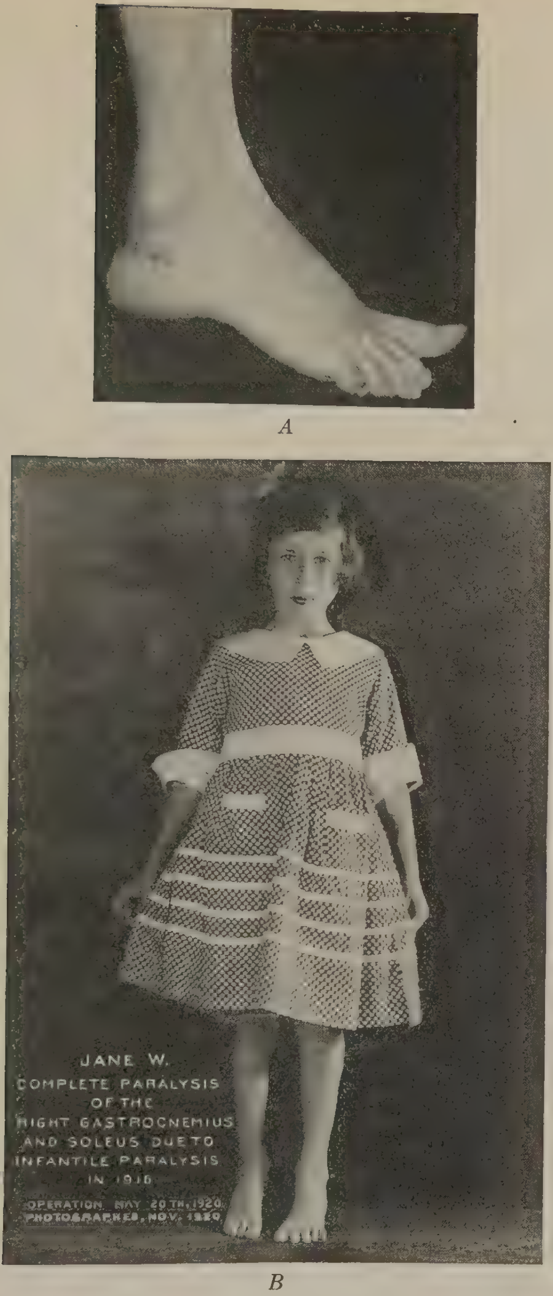

Fig. 63, *A* and *B*.—These photographs illustrate the effect of transferring the peroneus longus and flexor longus hallucis to replace the paralyzed gastrocnemius. and soleus. Before the operation there was a marked degree of cavus and total inability to hold the body-weight on the toes. Note the normal form of the foot and the ability of the patient to stand on tip-toe.

verse incisions are made over the back of the heel excising an ellipse the width of which is dependent on the amount of redundancy of the skin. Sufficient skin should be excised to draw the heel toward the knee with all the traction that the suture line can stand. Through the two lateral projections of this elliptical incision, the peroneal tendons are exposed on one side and the flexor longus hallucis tendon on the other.

The peronei are easily found but the flexor longus hallucis lies buried beneath a fascial plane which holds it close against the groove in the astragalus (Fig. 62). The large vessels and the posterior tibial nerve should be drawn forward until this fascial band is exposed and only after its incision will the tendon become visible. The tendons are cut off as low as possible, threaded with the usual fixation suture, drawn through a slit in the Achilles tendon and fastened firmly to the os calcis which has been grooved for their reception as in the previous operations.

The wound is closed by interrupted sutures, with sufficient tension to draw the tuberosity of the os calcis upward toward the knee. In putting on the plaster it is advisable to fix the heel in this position and after this first part of the plaster has hardened, to apply a second portion in which the forefoot is forced dorsalward so as to correct the cavus deformity (Fig. 63).

4. *Paralysis of the Extensors of the Knee.*—To correct this condition, transplantation of the ham-string muscles is done. In this operation the sheath method is not feasible since the tendons at the knee, unlike those at the foot, are not equipped with a sheath. This is due to the fact that with the exception of the quadriceps, the tendons run approximately a straight course and a sheath is a physiological necessity only when the tendons change their direction. In the case of the quadriceps tendon the knee joint and the quadriceps bursa take the place of the sheath. The technique of the operation, therefore, is quite different from that employed in the operations about the foot.

The site of implantation is first prepared by a longitudinal incision extending from a point 2 in. above the patella vertically downward to its lower pole. A small vertical incision is made from the upper border of the patella to about its middle and a corresponding horizontal incision along the superior border. Two little flaps are thus outlined which are retracted together with the superficial bone layers thus exposing the raw surface of the patella against which the implanted tendons are to be brought (Fig. 64). The second step of the operation consists in reefing the paralyzed quadriceps tendon by means of two mattress sutures. The purpose of this procedure is to pull the patella as far upward as possible so that when the transplanted muscles begin to contract, their full force will be exerted at once to extend the calf instead of expending part of their energy in first pulling the patella upward.

The patient is then turned on the side and a long incision is made exposing the biceps tendon from a point almost half way to the hip down to the head of the fibula. Before freeing the tendon, the external popliteal nerve must be found and drawn out of the way. In cutting away the tendon from its insertion, care must be taken not to injure the external lateral ligament of the knee joint, which, it will be remembered, also inserts into the head of the fibula. The tendon and muscle must be freed until its course, when transposed, will be an oblique one and not transverse. The more vertical the pull of the transplanted muscle, the more effective it will be and the sacrifice of a few muscle fibers is more than outbalanced by the increased mechanical efficiency secured by the proper line of traction. The tendon is drawn through a subcutaneous channel previously made by means of a stout dressing forceps. The channel must be sufficiently wide to allow free passage and the muscle itself must not be twisted during the transfer.

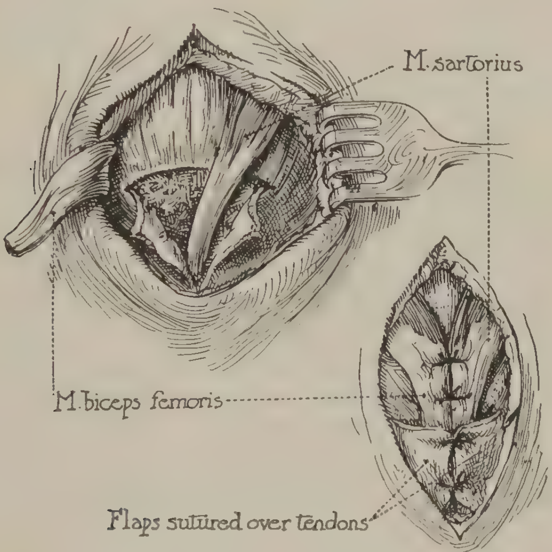

Fig. 64.—This illustrates the method of reflecting the osteoperisosteal flaps from the surface of the patella preparatory to the implantation of the hamstring tendons, and the suture of the osteoperiosteal flaps over the transplanted hamstring tendons after their attachment to the patella. The flaps hold the tendons firmly against the raw bone surface and insure firm union. The tendons are united to one another above the patella so as to make the line of traction as nearly vertical as possible. (From Mayer, Orthopedic Treatment of Gunshot Injuries.)

The biceps is not anchored to the patella at once, but before this is done, the patient is again turned so as to expose the inner side of the thigh and through a third incision, the inner group of ham-string muscles is exposed. Any one of these except the semi-membranosus, is suitable.

The operator selects whichever muscle seems most suitable, frees it a sufficient distance upward to make its course straight, and brings it forward through a subcutaneous channel similar to that already made for the biceps. The two transposed tendons are then cut off equal in length. Each is threaded with the usual fixation suture and then anchored securely to the patella in such a way as to rest against the raw bone-area already prepared for it. The retracted periosteal flaps are united over the tendons thus pressing them firmly against the bone (Fig. 24). Several stitches are taken above the implantation site holding the two tendons together. This renders the resultant pull almost a vertical one and thus approximates the action of the paralyzed muscle. The operation is concluded by the usual continuous skin sutures.

5. *Paralytic Drop-wrist.*—Transplantation of the pronator radii teres, flexor carpi radialis and flexor carpi ulnaris, to the dorsum of the hand. The operation is a complicated one and cannot be considered as reliable as those already outlined owing to the fact that the transplanted tendons are attached to paralyzed tendons instead of directly to the bone.

The first incision about 2 in. long, is made along the radial side of the arm just at the insertion of the pronator radii teres half way distant from the elbow to the wrist. The extensor carpi radialis longior and brevior are identified and drawn towards the ulnar side. This exposes the fibrous insertion of the pronator radii teres. Care is taken in freeing this from the bone, to preserve as much of the tendon as possible since it is otherwise too short. When this is freed it is drawn through a slit in the tendons of the extensor carpi radialis longior and brevior and fastened to them under tension by means of four or five silk sutures.

The second incision, also about two inches long, is made just above the annular ligament to the radial side of the common extensor tendon. The extensor communis digitorum and the extensor longus pollicis are identified and their tendons are severed 2 in. above the annular ligament.

The third incision, about 6 in. long, runs on the anterior surface of the forearm in the course of the flexor carpi radialis tendon. The tendon is freed to the point where it enters the bony canal in the trapezium and is drawn through a subcutaneous channel over to the mid line of the dorsum of the wrist. Its course should be as nearly vertical as possible. Through a slit in the flexor tendon about ½ in. from its extremity, the divided extensor tendons are drawn and fastened by fine silk sutures to the transplanted tendon (Fig. 65). The free end of the flexor tendon is implanted between the extensor tendons in such a way as to prevent its raw surface from coming in contact with the sheath. In performing this type of overlapping suture the apposing surfaces of the two tendons should always be thoroughly scraped so as to remove the gliding cells. The opposite sides of the tendons which come in contact with gliding tissue, should have the gliding cells carefully preserved.

The fourth incision exposes the flexor carpi ulnaris tendon. This incision is about 5 in. long. The tendon of this muscle is less adapted to the transfer than the flexor carpi radialis since its muscle fibers have a lower point of origin. In order to free it properly some of these lower muscle fibers have to be sacrificed. This tendon is also drawn to the dorsum of the hand through a subcutaneous channel and is attached to the extensor tendons superficial to the attachment of the flexor carpi radialis. The traction of the two flexor muscles results in a vertical motion almost identical with that of the paralyzed extensor. The hand is splinted in the hyperextended position with the fingers fully extended.

6. *Transference of the flexor sublimis tendon to replace the divided flexor tendons of an adjacent finger.*—This operation is indicated when, owing to extensive gunshot injuries, the destruction of the tendons is so great and the scar-tissue formation so dense as to prevent the union of the tendons by free transplantation. The results, although not perfect, have been satisfactory.

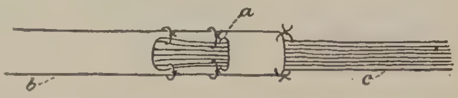

Fig. 65.—This diagram illustrates the suture of tendon to tendon when over-lapping is possible. Tendon *c* is drawn through a slit in tendon *b* and fastened to it by means of fine silk sutures.

The distal ends of the divided tendons are first exposed by means of a 1½ in. curving incision. The flexor profundus is identified and threaded with the Bunnell suture. The flexor sublimis tendon of the adjacent finger is next exposed by a 3 in. incision, the tendon cut away at a point sufficiently far distal to allow it to reach to the divided flexor profundus tendon. It is drawn downward beneath the skin to the injured tendon, threaded with the usual suture and united to it with just sufficient tension to render it taut when the finger is straightened out. The divided flexor sublimis is not sutured since this tends to impede rather than to assist the more important action of the flexor profundus.

7. *Transfer of the extensor communis digitorum of the index finger to replace the divided extensor tendon of the thumb, middle, ring or little finger.*—This operation corresponds to that already described for the flexor tendons. The indication is also given by extensive injuries in which the free transplantation of tendon is impossible because of dense scars adherent to the bone. The distal end of the divided tendon is first exposed and freed from the surrounding tissues for about ½ in., then a second incision is made along the extensor tendons of the index finger in their course over the metacarpal bone. The more superficial of the two tendons, namely, that of the extensor communis digitorum, is freed from the deeper tendon (extensor indicis). The tendon is cut off at a point sufficiently distant from the wrist to allow it to reach to the distal stump of the divided tendon. It is passed through a subcutaneous channel and fastened to the severed tendon by the overlapping method (Fig. 25).

(For the Steindler operation at the elbow see Chapter CXVI.)

Figure 66 illustrates the result of this operation performed on a patient whose extensor tendons of the middle and ring fingers had been destroyed by an electric burn.

After Treatment of Tendon Operations.—The importance of the after-care in cases of tendon transplantation cannot be too strongly emphasized. Even the best operation will prove a failure unless the transplanted tendons are properly exercised. The period of immobilization should not, in case of transplantation on the foot, exceed three weeks; for operation on the fingers, not more than two weeks. During the immobilization period the limb should be splinted in such a way as to relax all tension on the structures operated on. After the expiration of this preliminary immobilization, active motion of the muscles involved should be begun. Passive motion and massage is of comparatively little significance. It is, therefore, unwise to operate on children who are so young as to be unable to coöperate. In teaching a patient to use a tendon which has been operated on, a little ingenuity is required to bring out the desired result. Thus,

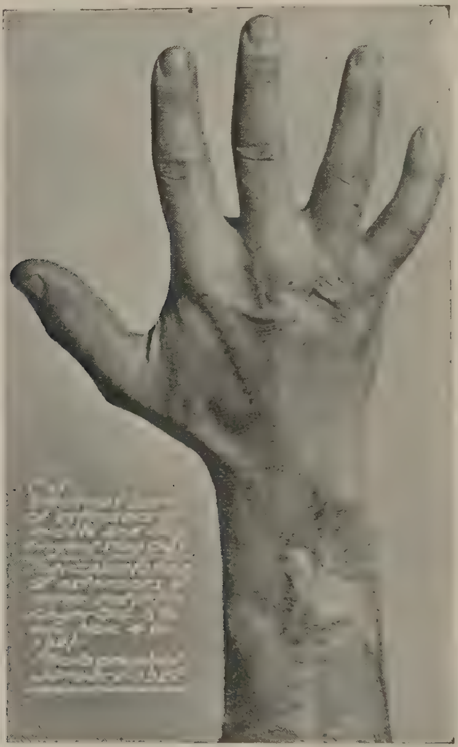

Fig. 66.

after the transfer of the peroneus longus for paralysis of the tibialis anticus, the tendency of the patient is to utilize the extensor longus digitorum instead of the transplanted muscle. The masseuse must teach the patient to relax the common extensor of the toes which is best done by asking him to lift the big toe, while the other toes are held flexed. In this way the inner border of the foot is raised without the everting action caused by the extensor longus digitorum. Children learn the trick very soon, when they are instructed correctly.

After transferring the biceps to replace a paralyzed quadriceps, the best preliminary motion is to ask the patient to pull the patella toward the hip. After this can be done, the patient is held in an upright position and the attempt is made to lift the leg forward with the knee extended. Within a very few weeks the patient will learn to use the transplanted ham-string muscles as extensors of the knee.

In working with the fingers, the problem is particularly hard since adhesions tend to develop very rapidly. The immobilization period must therefore be made shorter and consequently the healing process is not as complete. In addition to the suitable active exercises the use of little celluloid splints holding the finger at varying angles is helpful in overcoming the natural stiffness. Hot-water baths are also of some value, but the essential factor is the active coöperation of the patient in breaking down the adhesions, which, despite every refinement of technique, are almost certain to develop.

When the transplanted tendons have been taught to function, they still require the surgeon's attention, since their strength is almost never equivalent to that of the structures they are replacing. They must be guarded against undue strain by a suitable splint and further strengthened by systematic exercises.

Prognosis after Tendon Operations.—The prognosis in tendon operations depends almost altogether upon the type of operation. Certain operations should give almost 100 per cent. good results, others, not more than 50 per cent. To those giving a high percentage belong the tendon transplants on the foot in which either the peroneus longus or the tibialis anticus is shifted from one side to the other. The percentage of good results does not imply a perfect gliding of the transplanted tendon nor a perfect restitution of the function of the foot; it does mean that the transplanted tendon has sufficient voluntary motion to approximate the action of the paralyzed tendon, and that, though the normal strength of the foot is not regained, the muscle balance is restored.

In the third tendon transfer described for the foot (peroneus longus and flexor longus hallucis to replace the paralyzed gastrocnemius and soleus), a normal gliding of the tendons may be expected in all instances. The grave objection to the operation is that the strength of the paralyzed muscles is normally so much greater than that of the two replacing muscles, as to render a complete restoration of muscle balance impossible. The patient is however, enabled to rise on the toes and to walk with a scarcely perceptible limp; running and leaping is impossible.

The operation at the knee is not as perfect technically as the foot operations, but the functional results have been quite satisfactory. In all cases the patient is able to extend the paralyzed leg when standing and in some instances even when lying down. In all cases the gait is much improved.

The tendon transfers on the hand are still less certain procedures, since, instead of attaching the transplanted tendon to the bone, it must be fastened to another tendon. Though this type of union is never as firm as the bony, the period of immobilization may not be prolonged since, otherwise adhesions would develop. Despite this inherent difficulty, about 75 per cent. good results may be expected.

Of the free tendon grafts satisfactory function should not be expected in more than 75 per cent. Tendolysis may be expected to restore 50 to 85 per cent. of the normal motion of the finger. In these cases as well as in all finger operations, a great part of the success depends upon the coöperation and zeal of the patient, since it is only through the voluntary contraction of the affected muscle that the tendon can be made to glide. No form of passive motion or massage is of any avail. Electrical stimulation may help, but it never gives as great a range of motion as that produced by the voluntary effort of the patient.

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CHAPTER CX

TUBERCULOUS DISEASE OF THE BONES AND JOINTS

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TUBERCULOUS DISEASE OF THE SPINE

Tuberculous disease of the spine is a chronic inflammation with destruction of the bodies of the vertebræ, generally resulting in a posterior angular projection of the spinal column. This morbid condition is often called Pott's disease after Percival Pott of St. Bartholomew's Hospital, London, who, in 1779, first carefully studied and directed attention to the pathological condition. But not before 1836 was the condition recognized by Delpech and Nelaton as identical with tuberculosis of the lungs. The casual factor was not known until Robert Koch, in 1882, discovered the bacillus tuberculosis.

The origin and earliest history of tuberculous diseases of the bones are unknown, but we find indications of Pott's disease in the human remains from the Neolithic Period, five thousand years old. This diagnosis, however, needs confirmation and is merely suggestive. The oldest undoubted case of tuberculous disease of the spine is that described by Smith and Ruffer in an ancient Egyptian mummy (1000 B. C.) of a priest of Ammon. This individual was a young adult male and his mummy shows signs of Pott's disease in the form of a scoliosis of the lumbar region and a huge psoas abscess in the right pelvis. It is interesting to note the occurrence in Indian graves found in Tennessee and adjacent territory, numerous hunch-back images of clay which suggest pre-Columbian examples of Pott's disease, though the lesions found on the bones do not support this interpretation. Older lesions among fossil mammals, which lived before the appearance of man, have been described as suggesting tuberculous diseases of the bones, but this statement needs confirmation:*

Etiology.—(a) **Predisposition.**—*Age.*—Pott's disease is most frequent in early childhood between three and five years of age, though no age is exempt. Hayashi and Matsuke† found that in 700 cases of tuberculous spine 14.71 per cent. occurred between the ages of one and five years; 13.86 per cent. between 6 and 10 years of age: 17 per cent. between 21 and 25 years of age: 16.14 per cent. between 26 and 30 years of age. In their experience the greatest number occurred in the fourth year of life, then decreases from the fifth to the thirteenth, increases again steadily from the fourteenth to the twenty-first, maintains a high average from the twenty-second until the twenty-ninth, and finally decreases from the thirtieth to the sixty-fourth years of age.

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* Moodie, Roy L., The antiquity of Pott's disease and other spinal lesions: Primitive treatment. Surgical clinics of Chicago, iv, No. 3 (June, 1920), pp. 619–627.

† Hayashi and Matsuke, Zeitschr. F. Ortho. Chir. Bd. xxx, H3, 4, p. 381.

Sex.—Tuberculous spine disease is slightly more frequent in boys than in girls. Humphries and Durham* reported 517 cases of Pott's disease of which number 284 were boys and 233 were girls.

Localization.—Valtancoli's statistics,† based on 1004 cases of Pott's disease, give a very accurate localization of tuberculous spine disease, to wit: 42 per cent. involved the dorsal spine; 30 per cent. the lumbar region; 15.5 per cent. the lumbo-dorsal; 6.9 per cent. the cervical; 3.8 per cent. the cervico-dorsal; 1.8 per cent. the lumbosacral region.

Injury.—Opinions differ as to the rôle trauma plays in the causation of spinal tuberculosis. Obviously tuberculosis does not exist unless the tissues are invaded by the bacillus tuberculosis. Wilson and Rosenberger,‡ after experimental evidence, corroborated by clinical cases, concluded that trauma is merely a coincidental condition: On the other hand Valtancoli gives trauma as an etiological factor in 75 of his cases.

(**b**) **Infection.**—In surgical tuberculosis the exciting cause is the bacillus tuberculosis of either the bovine or human type. The relationship of these two types has been the subject of much investigation.

In 1911 the British Commission§ after an investigation lasting 10 years, reported that three groups of tubercle bacilli can be isolated in the tuberculous lesions of man. Group 1 has all the characteristics of the bovine type: Group 2 of the human type and Group 3 has some of the characteristics of both. Morphologically, bovine and human tubercle bacilli cannot be differentiated, but they are regarded as different types of the same organism. They differ in their cultural characteristics and in their power of producing disease in different animals. The bovine tubercle bacilli are virulent to rabbits in five or six weeks, after the occurrence of a generalized tuberculosis, whereas the human tubercle bacilli produce a tuberculous lesion in the lungs and kidneys which rarely cause death.

The Commission further reported that the bovine tubercle bacilli produce a large percentage of surgical tuberculosis, but rarely cause pulmonary tuberculosis. Fraser‖ studied the type of tubercle bacilli in 70 cases of bone and joint tuberculosis in children. The tuberculous material, obtained after operation, was injected into animals: Six weeks later the animals were killed. The differentiation of the organism was based upon five tests. In 41 cases the bovine tubercle was found: In 26 cases the human tubercle and in three cases the mixed type. The ages of the children were limited to 12 years and under, and these were separated into three groups. Group 1 was comprised of children under three years of age: Group 2 from three to six years and Group 3 from 6 to 12 years of age. In the first group there were 23 children in whom were found bovine tubercle bacilli, and in five the human tubercle bacilli. In the second group the bovine tubercle bacilli were found in nine and the human tubercle bacilli were found in 10 instances, while in the third group the bovine tubercle bacilli were found in nine and the human tubercle bacilli in 11.

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* Humphries and Durham, The Journal of American Medical Association (Jan. 27, 1917).

† Valtancoli, Chirurgia degli Organi di Movimento (April, 1921).

‡ New York Medical Journal (June, 1913).

§ The report of Royal English Commission of tuberculosis, x, No. 9 (1911).

‖ Fraser, J., (*a*) The type of tubercle bacilli in bone and joint tuberculosis, British Medical Journal, 1, 2728 (1913), p. 760 and Fraser J., (*b*) The type of tubercle bacilli in surgical tuberculosis of childhood, British Journal of Tuberculosis, viii (1914), p. 16.

Fraser then investigated the source of the milk supply to determine whether the child had been taking human or cow's milk. The human bacilli were found in 19 nursing infants and the bovine bacilli in four; while in children who had been drinking cow's milk, the bovine tubercle bacilli were found in 37 and the human bacilli in seven. The family history was next investigated, and in 21 cases there had been pulmonary tuberculosis in the immediate family of the child. The human tubercle bacilli were found in 15 children and the bovine tubercle bacilli in six.

Catteneo[*] after reviewing the literature on the identity of human and bovine tubercle bacilli, concluded that surgical tuberculosis reacted more frequently to the bovine type, while pulmonary tuberculosis reacted to the human type. Orth, J.,[†] believes that the bovine tubercle bacilli rarely occur in adults and only in about 10 per cent. of all tuberculous lesions in children. Raw[‡] states that bone tuberculosis is caused by the bovine tubercle bacillus conveyed by infected milk or meat. Incidentally, he mentions that in a number of foreign countries where children do not drink cow's milk surgical tuberculosis does not exist.

While these various investigations have definitely shown the importance of the bovine type of tubercle bacilli in bone and joint disease, and furnish a strong argument for a pure milk supply, it is unwise to assume that the human type of tubercle bacilli is a negligible factor.

Mode of Infection.—In the majority of cases, the most important focus of infection in Pott's disease is in the lymphatic glands, particularly the cervical, mesenteric and bronchial. The portals of entry of the tubercle bacilli are the tonsils, the oral and nasopharyngeal

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[*] Catteneo, C., The reaction of children to human and bovine tuberculosis, Ztsch. Kinderh., vl (1913), p. 506.

[†] Orth, J., The significance of the bovine tubercle bacilli for human beings, Berl. Klin. Wchnsch, xxx, lx (1913), p. 10.

[‡] Raw, N., Human bovine tuberculosis, Journal State Med. London, xxll (1914), p. 387.

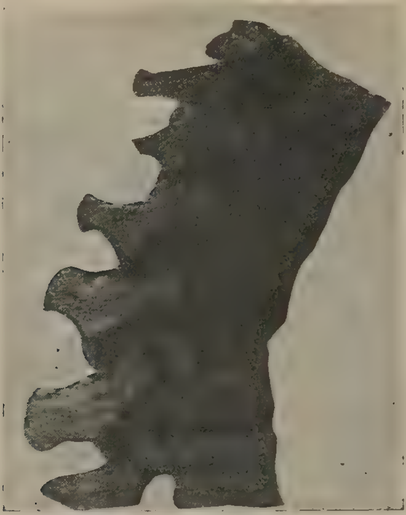

FIG. 67.—Showing involvement of the anterior portion of the vertebra with destruction of adjoining vertebræ and of the intervertebral discs. (From specimen in Warren Museum.)

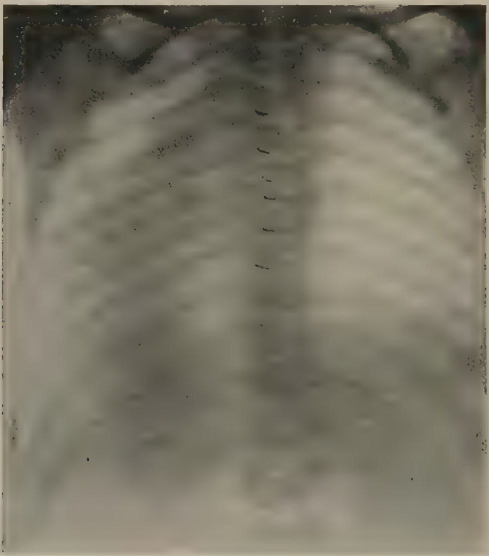

FIG. 68.—Roentgenogram, antero-posterior view, of an early case of Pott's disease involving the 6th and 7th thoracic vertebræ. Note that the bodies are in close apposition to each other.

cavities, and the digestive and respiratory tracts. As a result of the caseation and destruction of the glands mentioned, the bacilli gain access to the small veins and are carried to the right side of the heart, then through the systemic circulation and distributed to various parts of the body. Very frequently they may be destroyed by the proliferating endothelial cells as well as by the connective tissue cells and leukocytes, but if the resisting power of the child is enfeebled, then the bacilli take hold and a definite tuberculous lesion is the result.

Pathology.—The tuberculous process, as elsewhere, consists in the formation of tubercles and their caseation, followed either by fibrosis, healing or suppuration. The focus of infection usually occurs in the cancellous tissue of a vertebral body near the anterior surface. It slowly invades the cortex, periosteum, intervertebral discs and surrounding tissues. Occasionally the granulation tissue extends backward through the body of the vertebra and through the posterior spinal ligament, and involves the covering of the spinal cord producing pressure paralysis. Less frequently in children than in adults, compression of the cord is the result of an intraspinal abscess. The laminæ, the articular, transverse and spinous processes are seldom involved. In some instances the tuberculous infection appears just

Fig. 69.—Roentgenogram, showing marked deformity of the spine in a neglected case of Pott's disease.

beneath the anterior longitudinal ligament, spreads to the front of the spine following the course of the blood-vessels and involves the adjacent vertebral bodies. The intervertebral discs act as a check to the disease to a certain extent, but they soon become attacked and gradually disappear.

As a result of the tuberculous process the vertebral bodies are gradually undermined, lose their solidity and are pressed down or crushed by the superincumbent weight of the trunk (Figs. 67, 68).

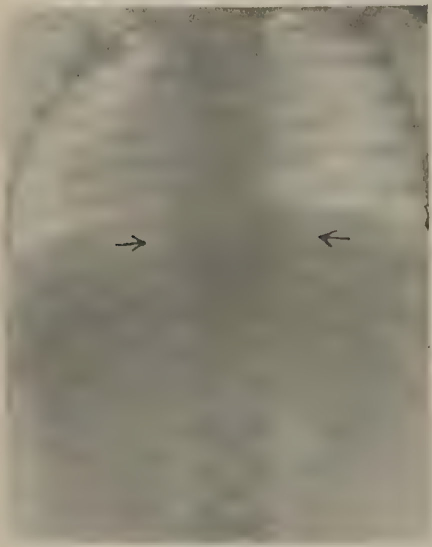

Fig. 70.—Tuberculous spine with abscess formation.

Fig. 71.—Roentgenogram, lateral view. Case of Pott's disease of three years' duration, showing marked destruction of the vertebræ.

This produces the characteristic deformity of the disease, namely, backward curvature, or kyphosis (Figs. 69, 71).

The deformity is sharply angular when only one vertebra is affected. When several contiguous vertebræ are diseased the deformity is more likely to be round, depending upon the number affected and the amount of bone destruction. The normal adjacent vertebræ above and below those diseased may become approximated, but they touch only with the anterior edges of their respective bodies, because the arches and lateral joints behind remain intact.

The granulation tissue thus having destroyed the bone trabeculæ, undergoes caseation and disintegrates into pus, forming a spinal abscess (Fig. 70). The contents may be liquid or semi-solid and may vary in color from pale-straw to dark-yellow. It consists of serum, fibrin, leukocytes and bony detritus, and the cavity itself is lined with a layer of granulation tissue. The abscess does not contain true pus unless pyogenic organisms have gained access.

Nature's method of repair in Pott's disease is ankylosis. The reparative process is affected by the diseased tissue being absorbed, then replaced by fibrous tissue which usually undergoes calcification and ossification. The vertebral bodies, in most instances, are the only portions of the spinal column that become ankylosed in Pott's disease, either by bone to bone contact, or by the intervention of fibrous tissue which may be converted into bone or less often into cartilage. Usually ankylosis is a combination of all three forms.

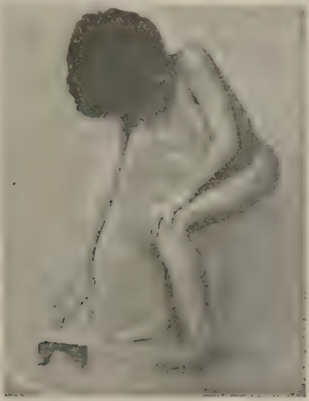

Fig. 72.—Patient picking up an object from the floor.

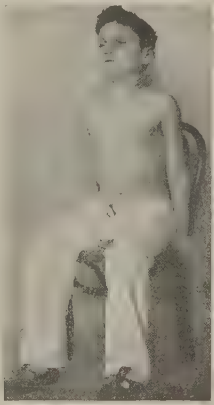

Fig. 73.—Characteristic sitting attitude assumed in dorso-lumbar Pott's disease.

Symptoms.—Tuberculous spine disease is characterized clinically by stiffness and awkwardness, by referred pain and by deformity of the spine. The symptoms are insidious in onset and only by careful physical examination can Pott's disease be detected at its inception. There generally is a history of fretfulness and a disinclination to play as actively as usual. The child walks with care, with the knees slightly flexed to avoid jarring the spine. When sitting or standing, unnatural attitudes are assumed such as putting the hands on the front of the thigh or resting the arms upon a chair or seat, or holding the head with the hands, depending upon the part of the spine affected (Figs. 72, 74). Lying on the abdomen is preferred to lying on the back. The attitude assumed is due to the effort on the part of the child to relieve the pressure on the superincumbent weight of the trunk and upper extremities on the diseased vertebral bodies. Pain is referred and is produced by pressure upon, or irritation of the roots of the nerves as they pass out of the intervertebral foramine. It varies with the location of the disease, but is more frequently abdominal. *A persistent pain in the abdomen in a child should lead to an examination of the dorsal spine for tuberculosis.* Direct pressure over the upper cervical vertebræ occasionally elicits severe pain, but if occurring in any other region of the spine, it militates against the diagnosis of Pott's disease.

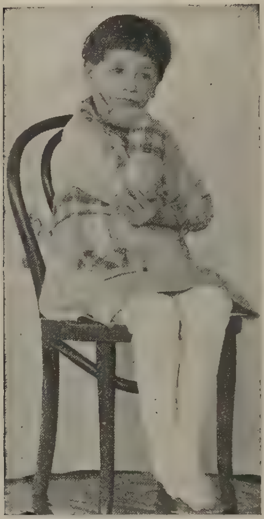

Fig. 74.—Characteristic attitude in cervical Pott's disease.

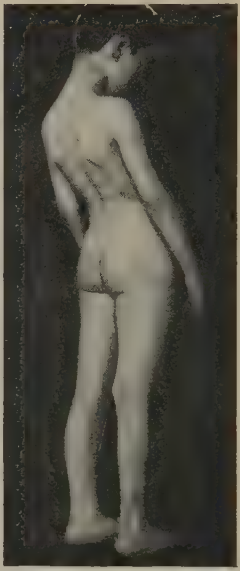

Fig. 75.—Early stage of lumbar Pott's disease showing marked muscular rigidity and abnormal lordosis.

Rigidity of the spine is the earliest symptom of Pott's disease. It results from muscular spasm and is of reflex origin (Fig. 75). It impairs function and is nature's method of fixing and immobilizing the diseased vertebræ. For instance, rigidity can be demonstrated in the dorso-lumbar region by placing the child on his abdomen, lifting the

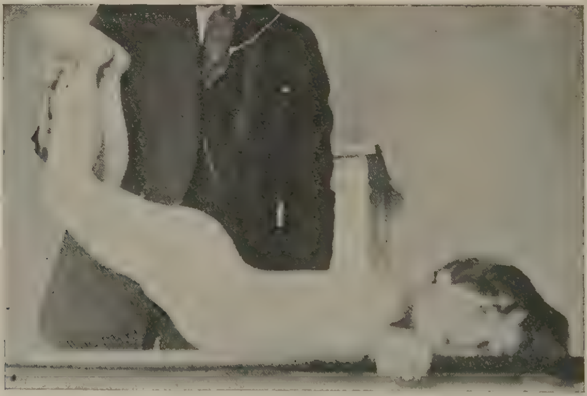

Fig. 76.—Illustrating the range of hyper-extension of a normal spine.

legs by the ankles from the table and also by moving them from side to side. Normally, by elevating the legs the dorso-lumbar spine can be hyper-extended to an angle of nearly 60° (Fig. 76) and lateral mobility to the extent of 30 or 40° to one or the other side of the median line; but in tuberculous spine disease, these movements cannot be made without including the thorax and dorsal spine (Fig. 77).

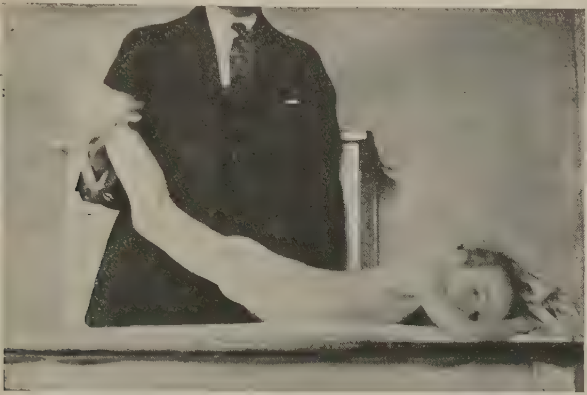

FIG. 77.—Illustrating the limitation of hyper-extension due to muscular rigidity.

Deformity of the spine occurs as a gradual projection backward of one or more of the spinous processes. Its appearance may be the first symptom for which advice is sought (Fig. 78). The size of the deformity depends upon the number of vertebræ diseased and the portion of the spine affected. In the early stages of the disease when only one or two of the vertebral bodies are involved, a sharp angular

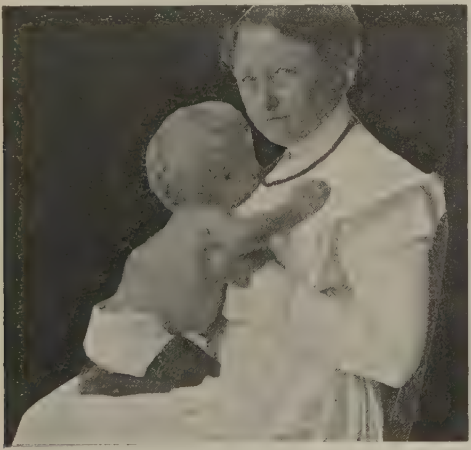

FIG. 78.—Pott's disease of the dorsal region, child two years old.

deformity (kyphos) may result, but as more of the vertebræ become involved, the deformity becomes larger and the angle less acute, and by the time a cure is perfected, the deformity has become more or less rounded. It is interesting to note that the appearance of the kyphos, whether sharp or rounded in character, often is of diagnostic value in that it indicates respectively the active or quiescent stage of the disease. The deformity is seen earliest and is more prominent in the dorsal, less so in the lumbar and least in the cervical region of the spine.

Fever is present in about 75 per cent. of all tuberculous spine diseases. If the course of the temperature be followed throughout the affection, there will be found an afternoon rise which varies from one-half to three degrees. In many of the apparently cured cases of tuberculous joint disease a slight but persistent afternoon rise remains and it is only a question of time until the patient will again appear with symptoms denoting a return of the original trouble. It is, therefore, important that a record of the temperature be kept from the beginning to the end of the treatment: The patient cannot be discharged as cured if there remains any rise of afternoon temperature, even though all other symptoms may have disappeared.

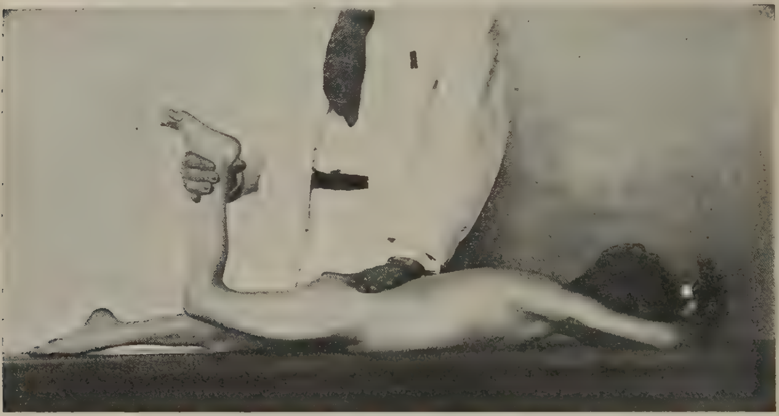

Fig. 79.—Examination for psoas contraction.

Secondary changes in the shape of the thorax are present in the more advanced cases of dorsal Pott's disease. Sometimes slight *lateral deviation* of the spine co-exists with a backward deformity, in which case the destructive process has extended to the side of the vertebral body.

The *complications* of Pott's disease are principally *abscess* and *paraplegia*. In early cases and under efficient treatment they are less likely to occur.

The signs and symptoms of tuberculous spine disease as mentioned above, are common to the disease in any part of the spine, but there is considerable variation in the different regions.

Cervical Region.—The earliest symptoms of cervical spondylitis are *pain*, neuralgic in character, referred to the neck and extending to the arms, and *rigidity* of the *neck muscles* which restricts the normal range of motion of the head. In looking upward or downward the child rolls the eyes as far as possible in either direction with little or no movement of the head, and when looking to the side, the shoulders and the trunk rotate in place of the cervical spine. If an attempt is made by the examiner to rotate the head, resistance is offered because of the apprehension of pain and of the muscular spasm. The head may be flexed and inclined to one side simulating wry neck if the upper two or three vertebræ are affected; if the disease is lower the head is inclined forward, and if still lower it is tilted upward and the shoulders are raised giving the appearance of a short neck. A favorite *attitude* is one in which the child sits with the hands supporting the chin (Fig. 74). There rarely is much deformity owing to the small size of the vertebræ and the shortness of the spinous processes.

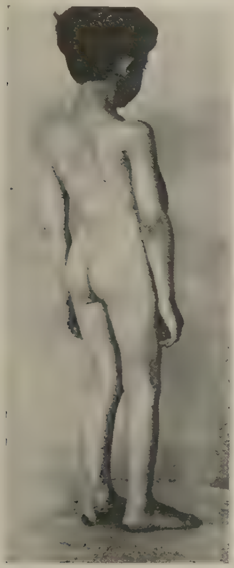

Fig. 80.—Tuberculosis of the dorso-lumbar region, showing deformity and characteristic attitude when standing.

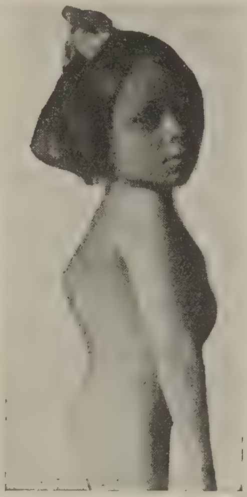

Fig. 81.—A neglected case of Pott's disease of the lower dorsal region.

Abscess is a very frequent complication in this region, and occasionally there is *paralysis*.

Dorsal Region.—The two upper dorsal vertebræ correspond in function to those of the cervical vertebræ and if involved by disease the symptoms will be almost identical with those of the cervical region already described, while the lower two vertebræ correspond in function to the lumbar vertebræ and if involved the symptoms will be similar to those resulting from a lesion of the lumbar vertebræ (Fig. 82).

Mid-dorsal Region.—In the mid-dorsal region the onset of the disease is insidious, usually the symptoms are not recognized before the appearance of the deformity which occurs early and quickly. Of importance is the history of *fretfulness*, of *irritability*, of a *disinclination to play* and of *pain* referred to the sides of the body and to the abdomen. The *attitude* is characteristic being that of over-erection with the shoulders well thrown back and the abdomen protruding. All the movements of the body are carefully guarded. The child often stands with the elbows resting upon a chair or table, and in sitting the hands grasp the seat of the chair, with the arms straight in order to support the superincumbent weight of the trunk (Fig. 73).

Grunting or labored respiration is pathognomonic of dorsal Pott's disease. This is due to the muscular rigidity restricting the normal movements of the ribs; inspiration is short while expiration is quick and is accompanied by a sudden closure of the larynx producing a grunt. This grunting respiration is more noticeable when the child is active. *Muscular rigidity* may be ascertained by testing the amount of backward and lateral bending of the spine in the manner previously mentioned (Fig. 77). This rigidity is of great diagnostic importance.

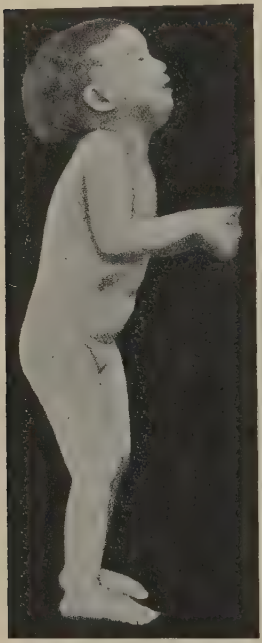

FIG. 82.—Showing characteristic attitude in disease of upper dorsal region.

Owing to the normal backward curve of the spine in this region the slightest degree of necrosis of the vertebral bodies produces a kyphos and hence *deformity* may be the earliest symptom (Figs. 78, 80). The kyphos may become very conspicuous even under careful treatment. Some compensatory lordosis occurs above and below the kyphos which makes the deformity appear even more prominent. In the early stage of the disease the kyphos is angular, then gradually becomes more or less rounded and generally larger by the time cure is affected. After the appearance of the kyphos all the symptoms mentioned above can be readily brought out by careful examination.

Deformity of the chest is particularly noticeable when the dorsal kyphos is prominent (Fig. 81). If the upper dorsal region is involved, then the sternum is pushed downward and forward while the ribs are held at a greater angle than normal. This puts the chest in a position of expiration. On the other hand when the disease is lower the chest is in a position of inspiration due to the raising of the sternum and ribs.

This distortion of the thorax interferes with the proper oxygenation of the blood in the lungs, producing malnutrition.

Paraplegia is a more frequent complication in dorsal Pott's disease than in the cervical or lumbar region. It usually is a late symptom, though it may occur at any time during the course of the disease, and even in the absence of a kyphos.

Lumbar Region.—*The carefulness of gait,* the *increased lordosis* or hollowness of the spine, together with marked protuberance of the abdomen, are especially characteristic of tuberculosis of this region. The child walks on its toes with the knees flexed to avoid jarring the spine. The increased lordosis is due partly to *muscular rigidity* and partly to the patient's efforts to relieve the pressure upon the diseased vertebral bodies. *Psoas contraction* (Fig. 79) too, when of mild degree may increase the lordosis, but when marked and unilateral it denotes abscess formation. The thigh is flexed resulting in a limp, and the posture is changed from over-erectness to forward bending to relieve the psoas muscular tension. *Pain* when present, is usually referred to the buttocks and thighs. Restlessness, crying and screaming may be present in the first hours of sleep. *Muscular rigidity* can be demonstrated not only by lifting the extremities and testing the backward and lateral bending of the spine, but also by observing the child's attempts to pick up an object from the floor. This is accomplished by flexing the knees and hips and gradually assuming a sitting or squatting position, but the trunk is kept in a rigid and erect position avoiding most carefully any movement of flexion. The child raises itself by pushing the body up, with the hands resting on the thighs and with arms extended (Fig. 85). *Deformity* does not develop in as early a stage as in dorsal or cervical regions of the spine.

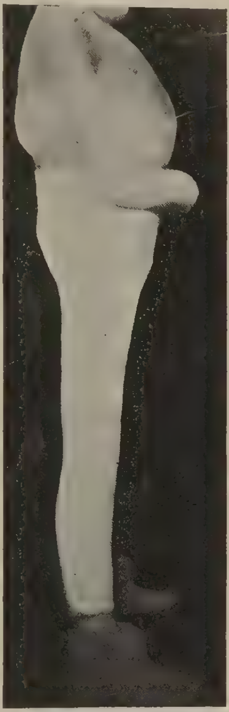

FIG. 83.—A case of arrested Pott's disease with a recent hip involvement.

Differential Diagnosis.—The recognition of tuberculous spine disease in its incipiency is of much importance because treatment instituted at the beginning of the disease may not only prevent or limit deformity, but shorten the period of treatment.

In the early stage of Pott's disease, when pain is referred to the arms, to the side of the chest, to the abdomen, to the buttocks, or to the hips, as previously described, many errors in diagnosis may arise. Rigidity of the spinal muscles, due to muscular spasm, resulting in stiffness and impairment of function of the spine, is the earliest symp-

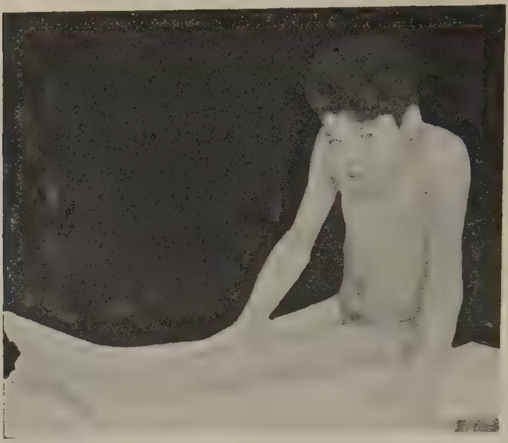

Fig. 84.—A neglected case of tuberculous spine involving the upper dorsal region.

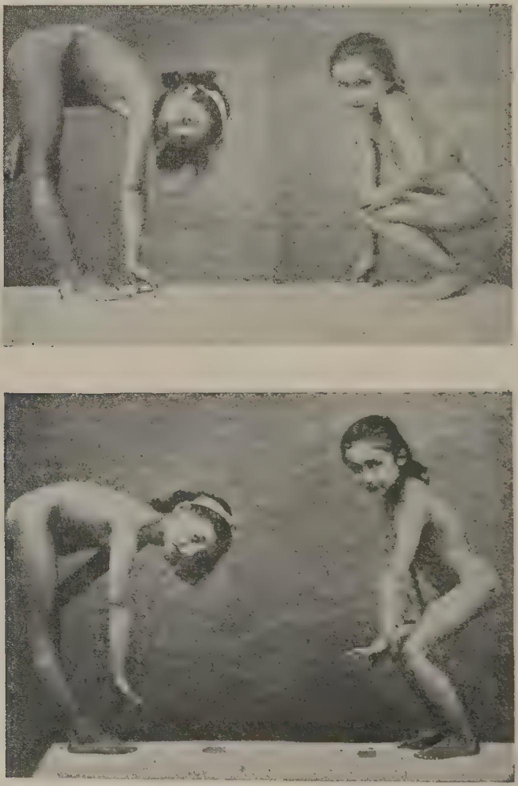

Fig. 85.—Illustrating the rigidity of the spinal column in vertebral caries. The children are picking objects from the floor. The contrast between the attitudes in health and disease are seen. (Wullstein.)

tom, and should lead to careful investigation. A sharp, angular, backward projection of the spine occurs early in tuberculous disease of the mid-dorsal region and the diagnosis is not difficult; but in disease of other regions of the spine the deformity is not developed as early and then the pathological condition may be overlooked. A roentgenogram may show no pathology and still does not disprove the existence of an incipient spine disease. In the initial stages it usually shows, in an anteroposterior view, disappearance of the space between the vertebral bodies (Fig. 68), and in later stages their collapse. In a lateral view, the destructive changes are more perceptible showing a crushed vertebral body or bodies and a backward curvature of the spine (Figs. 67, 68). The *von Pirquet cutaneous reaction* reveals tuberculosis past and present, but its use should be restricted to young children. A negative test is strong evidence against the probability of tuberculous infection.

Lumbar Pott's disease has been mistaken for tuberculous hip-joint disease, because of the referred pain to the hip and thigh, and on account of the flexion deformity of the limb due to psoas contraction producing a limp. In lumbar Pott's disease motion is restricted at the hip joint when hyperextending the limb; but it can be fully flexed and while in flexion, rotation is unrestricted. On the other hand in hip disease, *motion at the hip joint is restricted in all directions.*

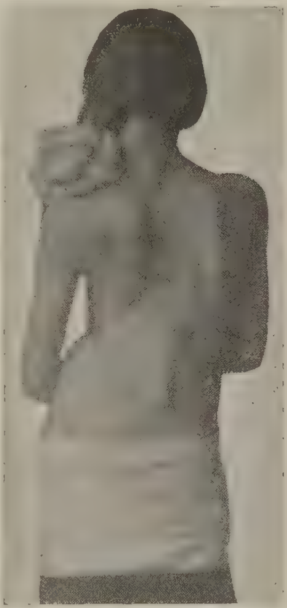

Fig. 86.—Tuberculosis of the lumbar region, associated with scoliosis.

Rachitic deformity is usually a rounded, backward curve of the dorso-lumbar region of the spine. It is neither as angular as in Pott's disease, nor is there as much muscular stiffness. When the child is placed face downward and the legs are elevated, a rachitic kyphos of mild degree will be changed into a lordosis. There is no pain or abscess formation and the deformity is generally associated with other manifestations of rickets.

Lumbago is uncommon in children. The onset is sudden with pain localized to the lumbar muscles. It is soon relieved under medical treatment.

Congenital torticollis may simulate cervical Pott's disease when there is lateral inclination and restriction of the rotary movement of the head. In torticollis the condition has existed from birth, the head is inclined towards and the chin points away from the prominent sternocleidomastoid muscle while motion is restricted in one direction only—away from the affected part. Then, too, the face is smaller on the affected side and the eyes are not on the same plane. In tuberculous spine disease motion of the head is restricted in all directions, the head and chin pointing in the same direction.

Scoliosis or lateral curvature is not likely to be mistaken for Pott's disease. On the contrary the slight lateral deviation which sometimes occurs in vertebral diseases may be mistaken for scoliosis, from which it may be differentiated by the absence of referred pain and muscular rigidity (Fig. 86).

Abscess pointing in the cervical or lumbar region, or in the groin, resulting from tuberculous spine disease, is sometimes difficult to differentiate from a similar collection of pus arising from other causes. (1) In *suppurating cervical glands* there may be general muscular spasm of the cervical region of the spine and sensitiveness on movement. Under such conditions it may not be possible to make a positive diagnosis until traction to the head has relieved the muscular tension: (2) *Perinephritic abscess* points in the lumbar region and can be distinguished from a cold abscess due to Pott's disease by the history of previous renal disease and the absence of spinal symptoms: (3) Appendiceal abscess may simulate a right psoas abscess of Pott's disease. It is recognized by the history of the case and by the character of the pus which is usually offensive, due to the presence of colon bacilli: (4) *Iliac abscess* due to a cellulitis arising from an extension of inflammation from the pelvic bones, is often difficult to differentiate from iliac abscess due to vertebral disease, except by the presence or absence of the symptoms of spine disease: (5) Abscess from a tuberculous hip usually gravitates and points at some place in the thigh, though occasionally it may travel up the sheath of the psoas muscle and point above Poupart's ligament. In addition signs of hip disease are present, particularly the limitation of motion in all directions of the hip joint.

Complications.—Abscess occurs in about 50 per cent. of all tuberculous spines, but only about one-half can be demonstrated clinically. Abscess is most frequent in the lumbar and dorso-lumbar regions, less frequent in the middle and upper dorsal region and least frequent in the cervical region. It appears slowly as a painless, fluctuating tumor on the surface of the thigh, abdomen, back, in the lateral region of the neck or in the pharynx. It may remain dormant for months and then gradually increase in size. The skin may become red and thin, and finally perforated. Air enters the cavity of the abscess and may spread septic organisms. When a sinus is established the abscess cavity continues to discharge indefinitely which is in part due to mixed infection. Amyloid degeneration, particularly of the kidneys and liver may be the ultimate result. In disease of the upper cervical region, the pus may push forward and appear under the mucous membrane of the pharynx as a **retropharyngeal abscess.** The symptoms are: Difficulty in swallowing, dyspnea and changed voice, best characterized as "nasal twang." The pus can be detected by the finger as a fluctuating mass in the pharynx. It is more common, however, for the pus to burrow through the deep fascia and appear either in front or in back of the sternomastoid muscle, or it may burrow into the mediastinum or pleura. In disease of the lower cervical vertebræ, the pus may burrow into the posterior mediastinum behind the esophagus. Abscess in this region rarely shows objective symptoms. When the upper and middle dorsal vertebræ are involved the pus may burrow between the posterior end of the ribs, following the posterior branches of the intercostal arteries and appear as a dorsal abscess, or the pus may burrow forward into the pleura, lung or mediastinum. Pus from the lower dorsal and sometimes from the middle dorsal vertebræ usually travels downward and enters the sheath of the psoas muscle (psoas abscess), and can be felt as a tumor in the iliac region. It may open above or below Poupart's ligament or even in the thigh.

A beginning psoas abscess causes characteristic symptoms even before it can be palpated. The thigh is slightly flexed and rotated inward, and the child leans toward the affected side. The method of testing for psoas abscess is to lay the child face downward on the table and to attempt hyper-extension of the thighs, one at a time, by flexing the leg to a right angle and then lifting it vertically from the table (Fig. 79). In a normal child the thigh can be hyper-extended about 15°, but this is not possible in psoas contraction.

In disease of the last two dorsal or the lumbar vertebræ, pus may make its way backward to the loin beneath the quadratus lumborum fascia and form just above the crest of the ilium posteriorly as a lumbar abscess. In some cases the pus burrows downward and backward and passes through the sacro-sciatic foramen and forms a tumor in the buttock; it may even spread along the course of the great sciatic nerve and descend to the posterior region of the thigh.

Paraplegia is not an uncommon complication of tuberculous spine disease in children; it is relatively frequent in adults. The liability to paraplegia is greatest in cases where the treatment has been inefficient.

There are two factors to be considered in discussing the morbid anatomy of Pott's paraplegia. First, pressure* from tuberculous granulation tissue which occurs mostly in children; and, second, pressure from intraspinal abscess which is usually seen in adults. Though other factors are sometimes mentioned, such as alteration in the position of the vertebræ producing an angular projection of the spinous process, or protruding sequestra at the side of the kyphos as a sequence of vertebral destruction, they cannot be seriously considered.

Paraplegia may appear with the very slightest or even with no deformity. It is not the bending of the vertebral column that causes the compression, but some encroachment on the lumen of the vertebral canal by the pathological process. In children the majority of cases of Pott's paraplegia is due to pressure upon the dura mater by extension of the tuberculous granulation tissue into the vertebral canal. This produces a disturbance of function without setting up a destructive or degenerative process. Only in exceptional cases does an intervertebral abscess occur. In adults, on the other hand, intervertebral abscess is the prominent factor in subjecting the spinal cord to pressure, and only occasionally is the pressure due entirely to granulation tissue.

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* Jacobs, Charles M., The Journal of American Medical Association (February, 1917), pp. 509–12.

The *histopathologic* picture of Pott's paraplegia is that of a *compression myelitis.* The tuberculous material breaks through the posterior portion of the vertebral bodies and through the posterior spinal ligament into the epidural space. It then invades the connective and adipose tissues outside the dura mater, which becomes thickened, covered by a deep layer of yellowish, caseous material, and by soft granulation tissue masses (peri-pachymeningitis). Eventually the tuberculous material presses upon the cord and the nerve roots. At first a moderate pressure is enough to excite functional disturbances without necessarily having an actual mechanical destruction of the nervous elements. If the pressure is protracted, severe damage to the cord occurs, resulting in so-called primary and secondary degeneration —even causing actual necrosis and severance of the cord.

The nerve roots may be subjected to pressure either in the spinal canal or as they pass through the intervertebral foramine, with consequent inflammation or atrophy.

Symptoms of Paraplegia.—Paraplegia may be the first clinical sign of an unsuspected Pott's disease, which can be unmasked by roentgenograms. The severity of the compression is correspondingly shown by the intensity of the clinical phenomena. The signs which signalize a threatening compression paralysis are in part motor and in part sensory. The most prominent local phenomena are those connected with the changes of motility; a weakness and stiffness of the limbs which impede the gait, usually first in one leg and then in the other. The gait is spastic, slow with short steps and dragging of the feet. The motor weakness gradually increases and may finally go on to complete paralysis. The tendon reflexes are invariably increased and the muscles of the lower extremities reach so great a degree of tonicity as to show the pronounced spastic type of paralysis (except in the lower lumbar region where the condition takes on a flaccid type): The limbs are then in a rigid tonic extension and can be flexed only with difficulty, owing to the muscular resistance. They show a very vigorous ankle clonus, exaggerated patellar reflex, adductor reflex, etc. Even before the paralytic phenomena appear, increased patellar reflex, ankle clonus, and the Babinski sign are present. If the seat of the morbid process is in the cervical vertebræ instead of the dorsal or lumbar vertebræ, then the arms are usually first and chiefly affected.

The cutaneous reflexes are usually retained or may be actively increased. In severe pressure paralysis, however, the cutaneous as well as the tendon reflexes may disappear. Disturbances of sensibility are often found independently of the referred pains due to the diseased vertebræ and can be definitely localized to determine the site of the lesion in the cord. Girdle sensation is very common. Disturbances of the bladder and bowels are often present—difficulty in micturition being an early symptom; later there is complete retention and finally incontinence.

Prognosis.—Tuberculosis of the spine is a curable disease. The average duration of treatment is four years when properly and efficiently treated. In the neglected or untreated class of patients the disease occasionally terminates in death, from tuberculosis of the meninges or lungs. Statistics do not give a correct idea of the percentage of mortality, because they are generally compiled from dispensary and hospital records. Social and economic conditions play a very important part in tuberculous joint disease. Children of the rich obtain purer air, more sunlight, more nutritious diet, more careful attention and more efficient orthopedic treatment than the children of the poor. Therefore, the mortality is more frequent in the latter class of patients, because hospital treatment is limited, and further care is taken up by dispensaries, which militates against a long-continued treatment so essential for a cure.

Septic infection following a tuberculous abscess leads to amyloid degeneration of the liver, spleen and kidneys, and is of grave import.

The occurrence of deformity varies according to the region of the spine affected, the amount of destructive process and the efficiency of treatment. The deformity is more liable to occur in the dorsal region than in the lumbar and is least frequent in the cervical region.

The vast majority of children with Pott's paraplegia recover under treatment with recumbency. Restoration of function usually follows recumbency in cases in which the stress has been moderate; for instance, when due to tuberculous granulation tissue which finally becomes absorbed. Occasionally the paraplegia may recur.

Treatment.—General hygienic measures are especially necessary and valuable. The *open-air* treatment is no less important in tuberculous infection of the spine than in pulmonary tuberculosis. Fresh air and sunlight are considered so essential for a cure that seaside and mountain sanitoriums, and numerous convalescent homes in the country are being established.

Sun Cure or Heliotherapy.—The exponent of this treatment is Rollier of Leysin, Switzerland. He cures surgical tuberculosis by solar radiation, most effective upon the high mountain slopes which offer the best climatic conditions. He believes that solar radiation has not only an extraordinary invigorating action, but is a most powerful bactericidal agent. The method consists in exposing the affected part to the sun for five minutes only, three times a day to avoid sun burning. Then the time is gradually increased so that at the end of a week it may last one and one-half hours. Gradually more surface is exposed and after several weeks of treatment the whole body has received the rays of the sun and has become walnut brown in color. In order to get the most benefit the sun bath is given for a period of four to six hours several times daily. Often during the early treatment the inflammatory symptoms are increased, but this is only temporary. Sunstroke and local aggravation of symptoms may occur if the exposure is too prolonged.

Kirsch and Gratz* have noted that old tuberculous sinuses frequently heal very rapidly under heliotherapy; that new sinuses frequently form but discharge only serous fluid and soon subside. Schrötter,† who has had much experience with heliotherapy states that the intensity of the radiation is less important than the length of the time of exposure and that treatment can be successfully carried out at a medium altitude or even at sea level. Alkan‡ brought to our attention that good results can be obtained by exposure to solar radiation on the *porches and roofs of city houses.*

Tuberculin.—The exact value and place of tuberculin in orthopedic practice is still unsettled. However, it is believed by most orthopedic surgeons that its use is not so promising in this particular field as it is elsewhere. Kleinberg§ reports 13 cases of bone and joint tuberculosis treated with increasing doses of tuberculin for a period of 7 to 26 months. Three of the cases improved, but two later developed a relapse; one was manifestly aggravated, while in all the other cases no benefit resulted.

Aside from the general measures enumerated, the treatment of Pott's disease, as in other tuberculous joints, resolves itself into the maintenance of rest for the diseased vertebræ, and the prevention of deformity, or, if already present, then the attempt to diminish it.

Conservative Treatment.—Conservative treatment means carrying out the principles of "physiological rest" either by recumbency on a Bradford frame, by casts, or by braces.

Recumbency.—Complete rest of the spine can be secured by removing the superincumbent weight and preventing motion. The simplest and most efficient method of accomplishing this is to place the child in the recumbent position upon a Bradford frame (Fig. 87) with or without a plaster cast. The frame is somewhat longer and slightly wider than the patient and is made of ½-in. gas pipe, over which a canvas is tightly laced, and over this a blanket and sheet. The child is secured to the frame by means of straps across the shoulders and by an abdominal binder and anklets (Fig. 88). In a few days the child becomes accustomed to the frame, which is then bent backward hyperextending the spine. This ordinarily relieves the pressure on the diseased vertebral bodies. But in disease of the cervical or upper dorsal region, traction to the head is also necessary, and this is accomplished by means of a chin sling attached to a weight and pulley at the head of the bed. Counter traction is maintained by the body-weight if the head of the bed is raised (Fig. 89).

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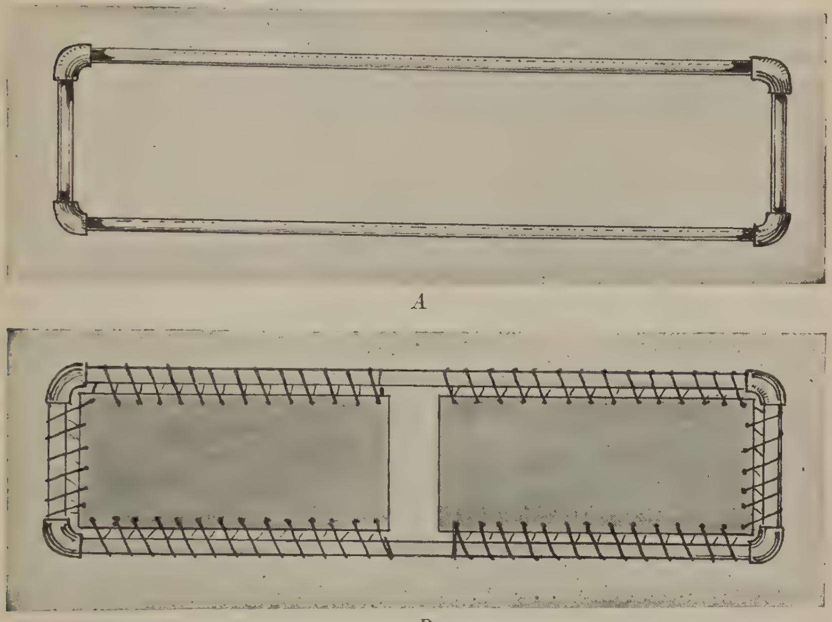
* Archiv. f. klin. Chir. civ., No. 2, p. 494.

† Med. klinik, li (1913).

‡ Berlin, Klin. Woch. (Aug. 4, 1913).

§ Kleinberg, S., The Journal of Orthopedic Surg. (December, 1919).


A

B

Fig. 87.—*A*, Bradford frame. *B*, Covered with canvas.

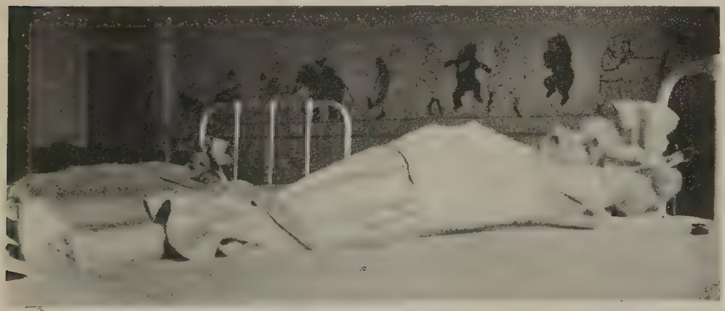


Fig. 88.—Recumbency on the Bradford frame with chin sling and anklets.

Recumbency on a Bradford frame is the treatment of selection. It improves the general nutrition, thereby increasing body-weight; it relieves pain and muscular spasm; it prevents or lessens deformity; it promotes abscess absorption; it relieves paralysis, and it aids in a cure by allowing ankylosis of the vertebral bodies.

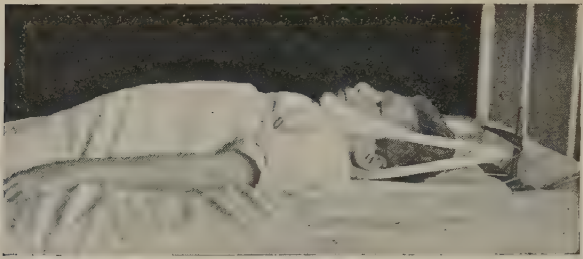


Fig. 89.—Head traction on Bradford frame in cervical disease.

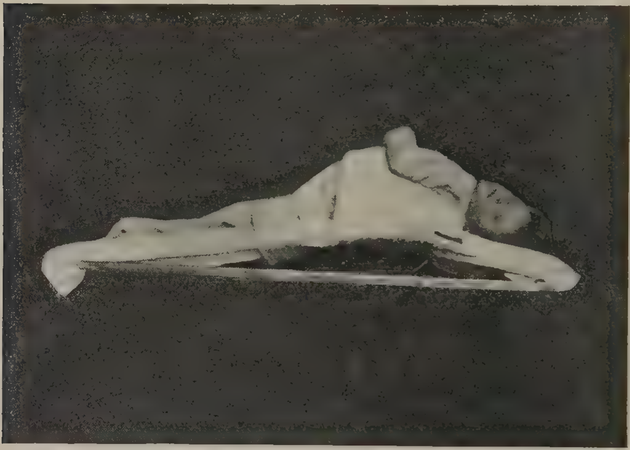


Fig. 90.—Bradford frame bent backward hyper-extending spine.

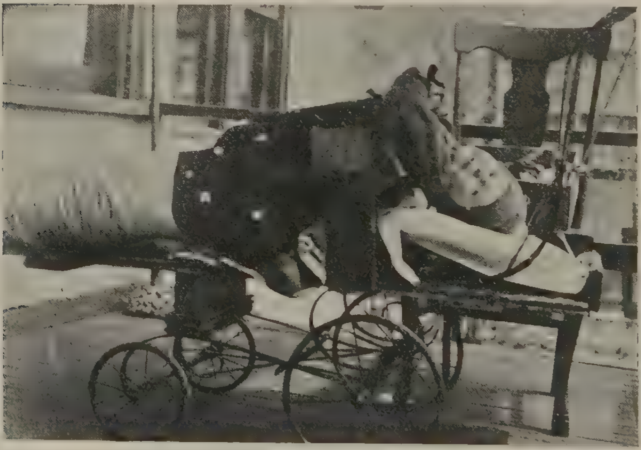


Fig. 91.—Portability of frame permitting open air and sunshine.

The recumbent position should be maintained from 12 to 18 months or until all active phenomena of disease, such as pain, abscess formation or paralysis have subsided. It does not preclude open air or sunshine, as the child with the frame can be carried from one place to another (Figs. 90, 91).

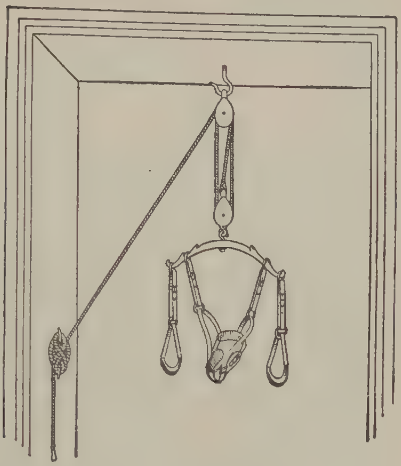


Fig. 92.—Sayre's suspension apparatus.

Plaster Jacket.—The plaster of Paris jacket is an anteroposterior support, and properly applied has added advantages not found in other supports. The jacket is usually applied with the child vertically suspended from the chin and occiput, or it may be applied with the child lying on a frame or hammock (Figs. 92–94).

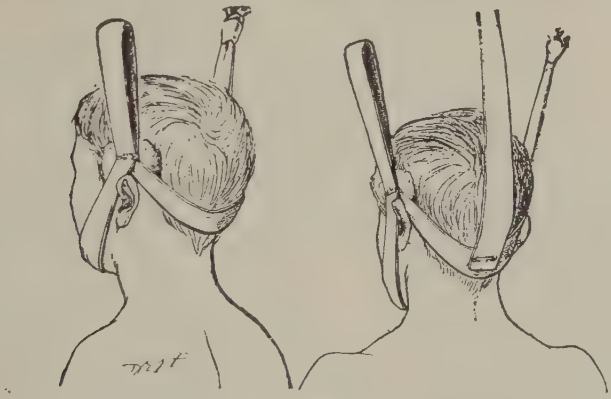


Fig. 93.—Calot's suspension apparatus. (Calot.)

Plaster Bandages.—Plaster bandages are made from crinoline, sized with starch, but not with glue. The crinoline is cut into strips, 5 to 6 in. in width by 3 yd. long, into which dental plaster is rubbed by hand. The bandages should be placed in a tin box or wrapped in paper and, in either case, kept in a warm, dry place ready for use. Commercial plaster bandages are useless.

In making a plaster jacket the bandages should be immersed on end in a full pail of warm water, one at a time as they are used, and left in until they have been thoroughly soaked, that is to say, until no further air bubbles rise to the surface of the water. The bandages are then taken out of the water and the two ends squeezed toward each other until they no longer drip.

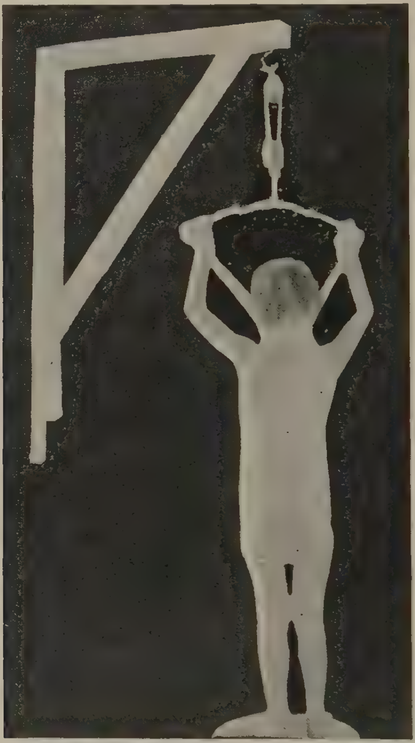


Fig. 94.—Vertical suspension by chin and occiput to extend the spine while body cast is being applied.

Application of the Plaster Jacket.—Before the plaster bandages are applied, a seamless stockinette shirting is put on the child reaching to the knees. If the head is to be supported the stockinette must be carried up and drawn over, an opening having been cut for the face. The child then stands on a stool and the halter of the suspension apparatus is adjusted to the chin and occiput; the arms are extended forward and held by an assistant or they are raised above the head and the end of the cross-bar grasped. Traction is made upon the rope

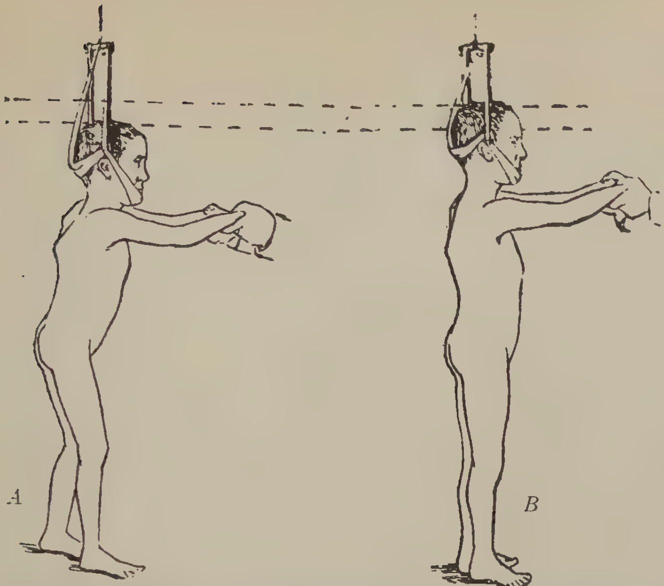


FIG. 95.—*A*, The cord has not been tightened. One sees in *B* that in pulling on the head, one has rectified the attitude and even corrected (slightly) the kyphos without the feet of the patient quitting the ground. (Calot.)

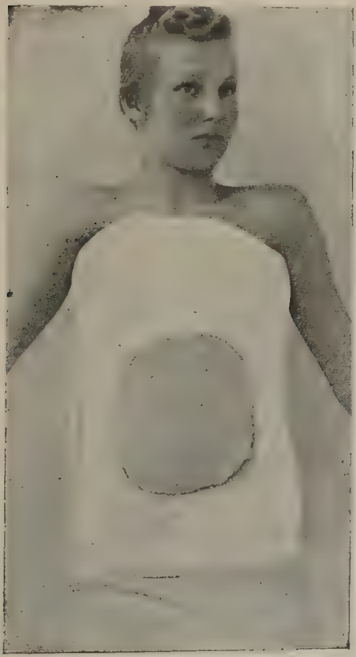


FIG. 96.—Plaster jacket with fenestration.

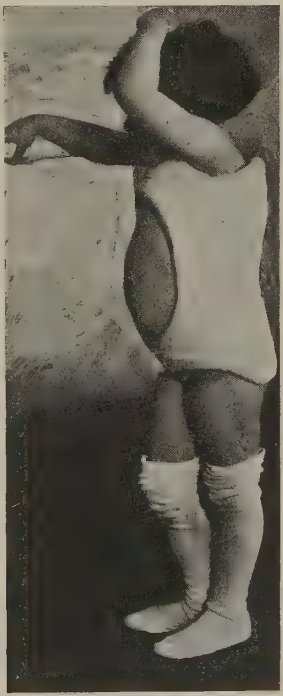


FIG. 97.—Plaster jacket with stockinette drawn over and sewed together.

just to the point where the heels are about to leave the stool. This extends the spine (Fig. 95). Strips of sheet-wadding are then wrapped around the stockinette, to protect the bony prominences from pressure. As an additional precaution against pressure on the spinous processes long strips of felt are placed longitudinally over the transverse processes. The "dinner-pad" is not essential.

The surgeon sits behind the patient, with an assistant in front, steadying the child's limbs between his knees. The bandages are applied with a smooth even pressure throughout each turn around the child's waist, and then downwards over the iliac crest to the greater trochanters, then upward to the upper margin of the sternum. Each layer should be rubbed with the next layer, so that they may adhere. The bandages are applied in this way until a uniform thickness of about ⅛ in. has been reached.

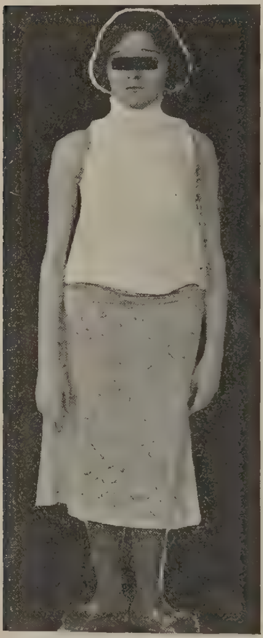


Fig. 98.—Body cast extending over shoulders and around neck for better leverage in upper dorsal Pott's disease.

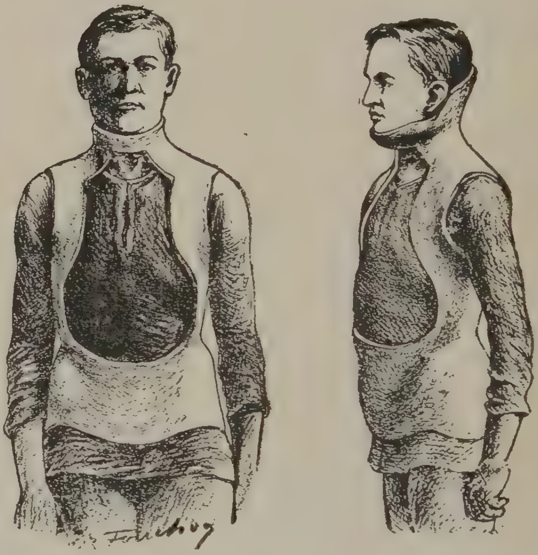


Fig. 99.—Increased leverage for upper dorsal or cervical disease. (Calot.)

When the plaster jacket has become firm it is trimmed, with a sharp knife, under the arms and at each groin, so that the child may sit with comfort. It is best to do this trimming while the child is in the recumbent posture. The lower and upper ends of the stockinette are drawn over the jacket and sewed together. After the application of the jacket the child should remain in bed 24 to 36 hours, as the jacket does not become absolutely dry before this time (Figs. 96, 97).

The height of the jacket depends upon the location of the disease. For disease below the tenth dorsal vertebra the jacket should extend in front, from the upper margin of the sternum to the groin, and behind, from the middle of the scapula to the buttocks. Better leverage will be obtained if the plaster bandages are carried up over the shoulders, uniting the front and back of the cast (Fig. 98). For disease above the tenth dorsal vertebra, the leverage can be increased by carrying the bandages up and including neck, chin and occiput, which have previously been protected by sheet wadding, leaving the face and upper part of the head exposed (Fig. 99). A fenestrum should be cut out from the cast over the kyphos, through which pads of felt are forced on either side of the spinous processes for the purpose of reducing the deformity.

A jacket need not be changed for two months, if it remains fairly clean and not uncomfortable. It is removed by cutting with a sharp knife. The line of removal should first be softened with water.

Steel Spinal Support.—At one time the Taylor brace was extensively used, but now various modifications have been designed. The principles are the same, however, namely, immobilization of the spine at the area of disease by leverage. Under certain conditions an adjustable spine brace is an indispensable means of treatment in Pott's disease, but its application presupposes great mechanical skill and experience on the part of the surgeon (Fig. 100).

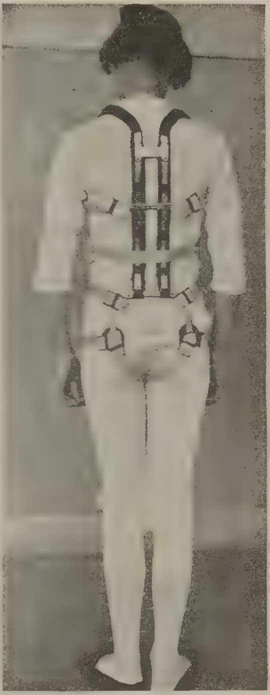


Fig. 100.—Taylor U-shaped spine brace.

If the disease is below the second lumbar vertebra, the steel brace is more effective than the plaster jacket, as it can be carried lower down and holds the pelvis with greater security. In the final stage of Pott's disease, the brace should be substituted for the plaster cast. It offers protection and allows tracings to be made of the spine and in this way keeps a record of the deformity. It is only possible to determine that sufficient cicatrization of the diseased vertebra has taken place when the deformity has remained stationary for years.

Treatment of Abscess.—Abscess formation complicating tuberculous spine disease, is best treated expectantly. In some cases the abscess will disappear without any special treatment while in a very large number it will subside with treatment merely by recumbency. If, however, the abscess does not subside, but increases in size, the skin gradually becomes red, thin and eventually necrotic. In such conditions it is advisable to aspirate or incise the abscess to prevent necrosis and spontaneous rupture. Neither aspiration nor incision is of any curative value. The abscess will be diminished in size by whatever method employed, but generally will refill. Aspiration rarely succeeds in emptying the sac, because of the caseous masses present. For this reason incision is preferable. It must be done under the strictest aseptic precautions. After expressing the contents of the sac, the incision is closed with sutures without drainage, and a dry sterile dressing is firmly applied.

Incision is indicated also in abscess formation which threatens important structures or interferes with effective immobilization; for the relief of pain due to the distension of the tissues, and in severe septic absorption resulting from the suppuration.

In retropharyngeal abscess, incision is advisable only in case of severe serious dyspnea. If incision is made in the middle line of the pharynx, then the child should be lying down with the head lower than the rest of the body, so as not to allow the pus to flood the larynx. In this instance the incision is left open.

Bismuth Paste.—Experience at the Home for Destitute Crippled Children, Chicago, has shown that after cold abscesses have been opened, evacuated and filled with bismuth paste, they would apparently heal in 13 to 21 days, but later refill with pus, and open spontaneously. This led to the abandonment of the paste in the treatment of tuberculous abscesses, when the disease was still active.

Treatment of Psoas Contraction.—The most efficient means of overcoming psoas contraction is to place the child on a gas-pipe frame and apply traction to the limb in the line of deformity by means of weight and pulley. A more rapid, though less safe, method is to anesthetize the child and straighten the limb by moderate manual traction and leverage; a plaster cast is applied to retain the limb in the desired position. In neglected cases the flexion of the limb usually persists and becomes fixed, as a result of the structural shortening of the muscles. Flexion, then, can be overcome only by tenotomies, followed by manual traction of the limb. A cast is then applied.

Treatment of Compression Paralysis.—Complete rest on a Bradford frame is especially indicated in the treatment of compression paralysis. In addition thereto, fixation of the spine by a plaster cast is advisable. If the disease is in the cervical or upper dorsal region, traction to the head by means of a chin sling is essential (Fig. 89). The recumbent position must be maintained from 12 to 18 months, or even longer. The first visible signs of improvement are the voluntary movements of the toes which is followed by gradual restoration of the muscular powers.

Osteoplastic Treatment.—The success of operative treatment for Pott's disease by inducing fixation of the spine has become definitely established. The two types of operations generally performed are those associated with the names of Hibbs* and Albee.† Hibbs induces fixation by "fusion" of the spinous processes, laminæ and lateral articulation; while Albee transplants into the spinous processes of the vertebræ an autogenous bone splint taken from the tibia.

Undoubtedly, both the Hibbs and Albee operations often promise a far more rapid cure than the conservative methods. They afford early relief from discomfort and pain, and they may shorten the period of protective treatment, such as is given by casts or braces. Nevertheless, operative procedure should not be the treatment of routine but of election, and the determining factors should be age, environment and special conditions.

In children, osteoplastic surgery should be reserved for cases in which it is either impossible to properly carry out conservative treatment, or after a trial it is found impossible to maintain control. In adults, time is an important sociologic and economic factor and any procedure which can hasten a cure should be employed. Both the Hibbs and Albee operations, to be of lasting result, should be followed by mechanical support of the spine for an indefinite period in accordance with the progress of the case (Fig. 101).

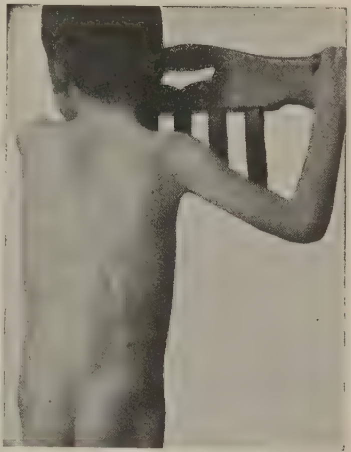


FIG. 101.—Six months after bone graft operation for lumbar Pott's disease.

TUBERCULOUS DISEASE OF THE HIP JOINT

Tuberculous hip-joint disease is a chronic destructive process, resulting in loss of function and deformity. It ranks next in frequency and importance to tuberculous spine disease. It is synonymous with hip disease, tuberculous arthritis of the hip, tuberculous coxitis and morbus coxæ.

The etiology in general has been discussed in the section on tuberculous spine disease.

Pathology.—The pathological process usually begins in the upper part of the femur, less frequently in the acetabulum and sometimes, primarily, occurs in the synovial membrane. Kepler and Erkers,‡ in

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* Hibbs, R. A., New York Medical Journal (May, 1911). Journal Am. Med. Assoc. (Jan. 20, 1917).

† Albee, F. H., Journal Am. Med. Assoc. (September, 1911).

‡ Arch. F. Klin., Chir., Heft 3 (1914).

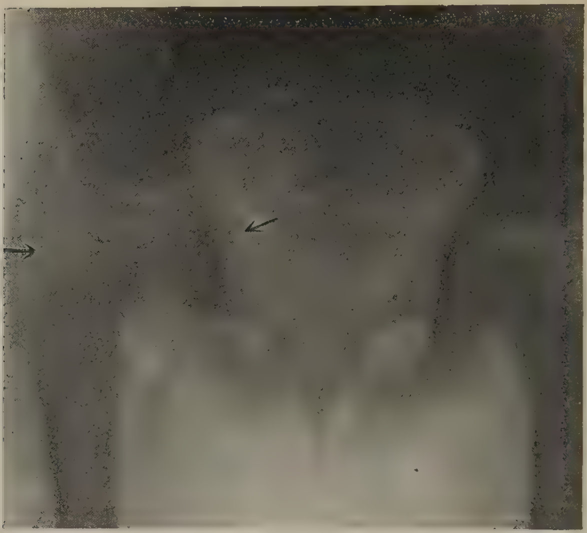

Fig. 102.—*X*-ray showing early stage of hip disease (right).

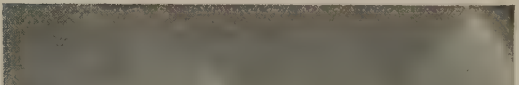

Fig. 103.—Early hip disease involving the femoral neck.

235 cases, found the primary focus in the femoral head 80 per cent.; in the femoral neck 8 per cent.; and in the acetabulum 12 per cent.

In the early stage of the disease, the joint shows signs of "sympathetic irritation," with increase of fluid within the joint. As the disease progresses the synovial membrane becomes thickened, granular, hyperemic and adhesions occur within its folds: The head of the femur is worm eaten in appearance, and the fluid within the joint may be of sufficient quantity to be interpreted as a tuberculous abscess

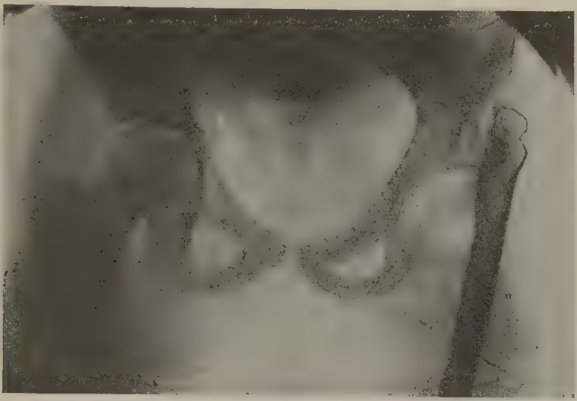

Fig. 104.—Neglected case of hip joint disease, showing destruction of the femoral head with upward displacement.

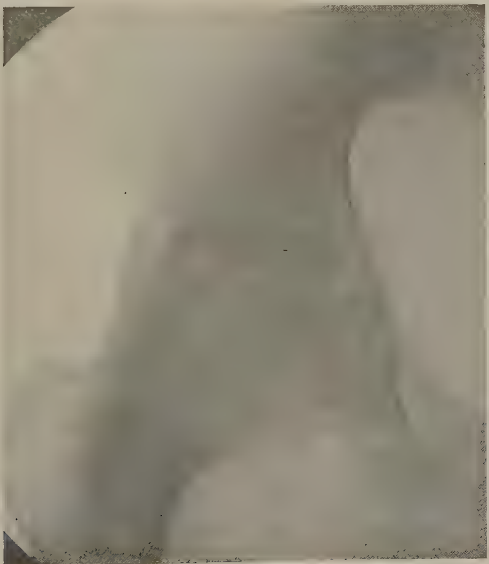

Fig. 105.—Cured case of hip disease with bony ankylosis.

(Figs. 102, 103). The abscess usually perforates the capsule and escapes into the tissues of the thigh, but in certain instances the acetabulum may be perforated and the tuberculous pus enters the pelvis.

After the tuberculous process has existed for a considerable time, the following changes take place: The femoral head is eroded; the cartilage is loose, yellow and necrotic, and lies in sero-purulent fluid. The acetabulum, usually involved secondarily, is generally roughened, eroded and softened. As a result of pressure and friction on the softened bone, its cavity is enlarged upward and backward, forming a "wandering" acetabulum. The capsular ligament is softened and relaxed. The femoral head may be destroyed and the femur displaced upward.

The end results are fibrous contractures, adhesions, and bony ankylosis (Figs. 104, 105). There is some muscular atrophy and general retardation of growth of the limb.

Symptoms.—The symptoms that first attract attention to the hip disease are a limp and pain. Of the two, the *limp* is the more important. It is a very constant symptom, remaining until, and frequently after a cure has been effected. It is not only due to sensitiveness of the joint, but also to interference with the function of the limb; for instance by the patient's inability to fully extend the thigh—the result of reflex muscular spasm.

Pain is more often referred to the anterior and inner side of the knee, and, when persistent, is misleading and often mistaken for knee-joint trouble. When filaments from the anterior crural, sciatic and obturator nerves, supplying the joint and surrounding parts are irritated by the local process, pain occurs. It may disappear long before the disease is cured. Pain at night, commonly called "night cries," is an important symptom. The child is restless in sleep and suddenly gives a frightened scream. The explanation offered is that the voluntary muscles protecting the joint relax, thus allowing sudden movement to take place which irritates the sensitive joint.

The other symptoms of hip-joint disease are elicited only by physical examination.

Inspection reveals *flexion deformity*, that is, flexion of the thigh on the pelvis—the result of reflex muscular spasm, with or without *abduction* or *adduction*. Flexion lessens the direct jar upon the sensitive joint (Figs. 106, 107).

Abduction usually is accompanied by outward rotation of the limb, and acts as a "*prop*" instead of aiding in the propulsion of the body (Fig. 108).

Adduction accompanies inward rotation, and is the attitude assumed for the protection of the sensitive joint (Fig. 109).

Flexion, abduction and outward rotation is the attitude of normal limbs when at rest but this passive attitude is seen early in hip disease, presenting an apparent or false lengthening of the limb. In order to bring the limb in a position parallel with the opposite one, the pelvis must be tilted downward on the affected side. As the disease progresses and destructive changes occur in the joint, the attitude of the limb

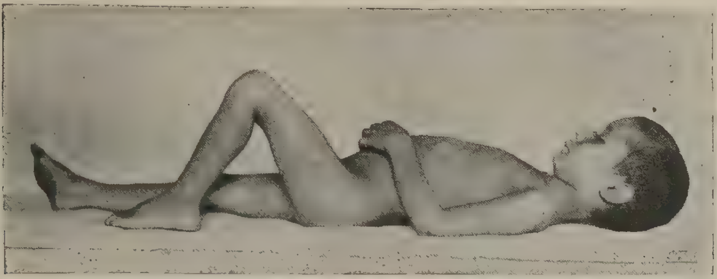

Fig. 106.—Flexion deformity to nearly a right angle.

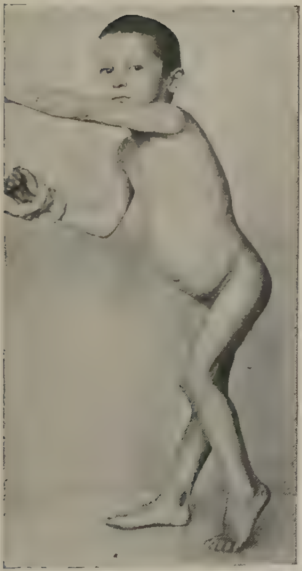

Fig. 107.—Same patient standing.

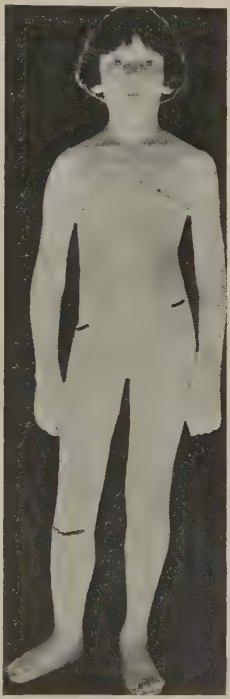

Fig. 108.—Attitude assumed when standing in an early stage of hip disease. Abduction of the right limb. Pelvis is tilted downward on the side of the affected limb.

changes to one of increased flexion, adduction and inward rotation presenting an apparent or *false shortening of the limb* (Fig. 110). For the purpose of bringing the limb into a position parallel with the opposite one, the pelvis must be tilted upward on the diseased side and downward on the other resulting in *apparent* or *false shortening*.

Muscular atrophy is a constant and very valuable symptom. It appears early and persists throughout the course of the disease. There has been much discussion as to the cause. The following theories have been given: (1) Reflex origin, due to irritation by the local process of the nerve filaments supplying the joint; (2) involvement of the nerves whose filaments supply the joint and muscles—a neuritis; (3) diminution in size of the femoral artery and its main branches, which have been held responsible for the trophic disturbance in the limb by reason of the decreased blood supply; (4) functional disuse, as a result of fixation in the treatment of the disease.

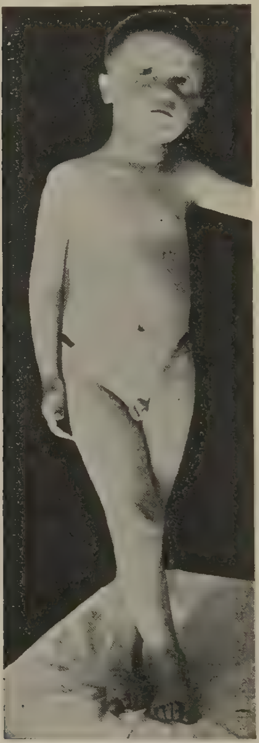

FIG. 109.—Apparent shortening. In order to bring the anteriorsuperior spines on the same plane the right leg is crossed over the left which plainly shows the amount of adduction deformity.

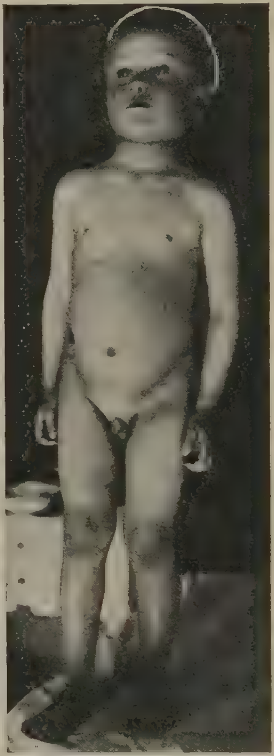

FIG. 110.—Apparent shortening of the limb. Note that the right pelvis is on a higher plane than the left.

The muscular atrophy corresponds to the duration of the disease, and the consequent disuse of the entire limb. It occurs first in the thigh, then in the buttock, and finally in the calf of the leg. The amount of muscular atrophy of the thigh and calf can be ascertained by measuring the circumference of the limbs at an equal distance from the anterosuperior spine.

Muscular spasm is by far the most important symptom of hip-joint disease, being the first symptom to appear and the last to disappear. *It restricts motion in all directions.* Upon this symptom alone one may base a diagnosis of hip disease; without it a positive diagnosis is not justifiable, notwithstanding the presence of other clinical findings.

Hip disease is differentiated from other diseases which simulate it by the presence or absence of muscular spasm, which is of reflex origin, the result of irritation of the anterior crural, sciatic and obturator nerves supplying the joint.

In examining for muscular spasm, the sensation imparted to the hand of the examiner is that of a sudden and positive stop. The test is best made with the child lying on a hard surface, as a table. Motion is usually tested for flexion, abduction, adduction and hyper-extension, in the order named. The limb is held at the ankle or knee with one hand, while the other one steadies the pelvis. The limit to the amount of flexion is determined by gently raising the limb until it comes to a positive stop, but if further attempt is made to force the limb higher, then movement at the pelvis begins. Limitation of abduction or adduction is determined by gently abducting or adducting the limb, and if limitation is present the pelvis moves with it. The test for limitation of hyper-extension is made by examining the child lying upon the abdomen then with one hand placed on the sacrum, the other one alternately grasps the ankle and raises the thigh from the surface of the table, the diseased limb cannot be raised to the same extent as the other without moving the sacrum. It is best to begin the test with the well limb in order to compare the normal movement possible at the hip joint.

False or apparent shortening is indicative of adduction deformity, and may co-exist with true shortening. It is measured from the umbilicus to the internal malleolus and the difference between the two sides gives the amount of false shortening.

True or actual shortening is a common symptom in the later stages of hip trouble, and may be due to any of the following causes: (1) Arrested growth, the result of disease of the epiphysis of the head of the femur: (2) Disuse of the limb throughout a long course of treatment: (3) Upward displacement depending upon bone destruction, such as erosion of the head of the femur and the upper border of the acetabulum, or upon fracture of the weakened neck of the femur. Actual shortening is the difference between the two limbs when measured from the anterosuperior spine of the ilium to the inner malleolus.

Fever is observed in about 75 per cent. of tuberculous hip disease. The course of the temperature runs almost identically with that described in the section on spinal disease. Here, too, one can see the temperature rise every afternoon anywhere to 100 to 102°F. in the early stage. Gradually the temperature decreases, but if it persists afternoons, no matter how slight the rise may be, a cure has not been accomplished, exacerbations being certain to recur sooner or later.

The record of the temperature, representing as it does, a reliable prognostic index, should be kept with conscientious care while the patient is being treated. Complete cure means not only a cessation of the symptoms but a permanent normality of the body temperature, with special reference to the afternoon period.

Tuberculous abscess is the product of the tuberculous process and is a very common complication occurring in about one-half of all cases of tuberculous hips. An acute exacerbation of symptoms, particularly pain and elevation of temperature, suggest abscess formation; in time a swelling may be observed, tender to pressure. Heat and redness of the skin are present only when the abscess is about to break. The most usual situation for the abscess to point is a little in front of and internal to the greater trochanter. Less frequently it may open in the gluteal region, or on the inner side of the femoral vessels below Poupart's ligament (Fig. 111).

A diagnosis of hip disease may frequently be made by what is known as the *Thomas flexion test.* It consists of flexing the normal thigh on the chest, which fixes the pelvis and straightens the spine. If hip disease is present the affected thigh is held at an angle because it cannot be in an extended position without producing lordosis of the spine (Figs. 112, 113).

The Differential Diagnosis.—Tuberculous hip disease is to be differentiated from *traumatism, rheumatism, lumbar spondylitis, Perthes' disease, sacro-iliac disease, congenital and traumatic dislocation of the hip, fracture of the femoral neck, coxa vara and sarcoma.*

Traumatism of the hip in young children frequently produces a condition closely resembling hip disease. It can be distinguished only by continual and painstaking observation.

Rheumatism is usually of sudden onset, and is more often preceded by repeated attacks; pain is more acute and *motion is not restricted in all directions as in hip disease.* Treatment with antirheumatic remedies relieves the condition.

Lumbar spondylitis in the early stage, before the deformity, when pain is referred to the hips or thighs and psoas contraction causes a limp, resembles hip disease. In lumbar Pott's disease psoas contraction restricts only extension and rotation during extension; motion is free in flexion and in rotation during flexion. In hip disease all *movements at the joint are restricted.*

Perthes' Disease (Osteochondritis) may simulate hip disease by early limp. Motion at the hip joint is principally limited in abduction and to a slight extent in internal and external rotation; all other movements are free. Pain is a negligible factor. Roentgenograms show a flattening or crushing of the upper epiphysis of the femur (Fig. 115).

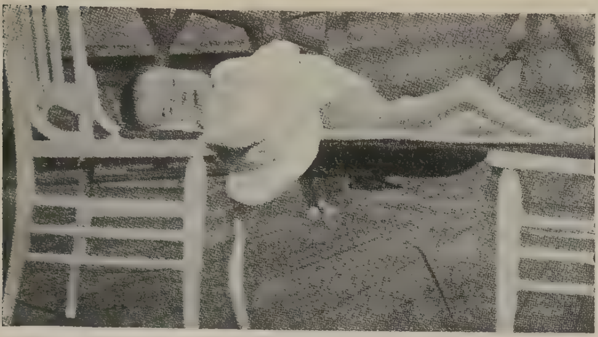

FIG. 111.—Flexion deformity, muscular atrophy and cold abscess formation.

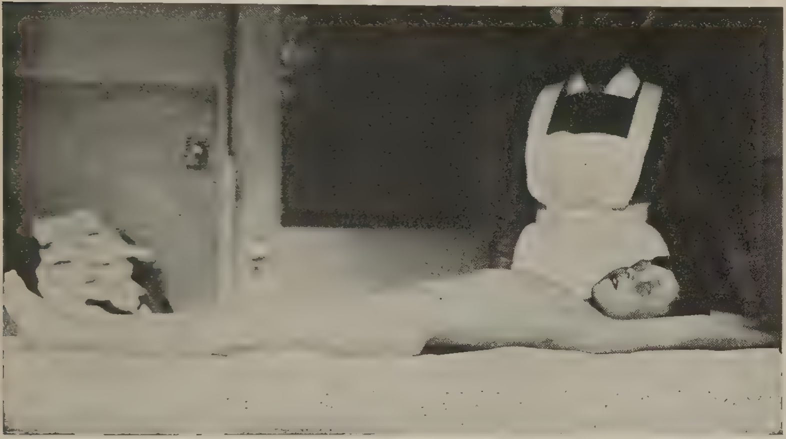

FIG. 112.—Note the lordosis of the spine when the affected limb is extended.

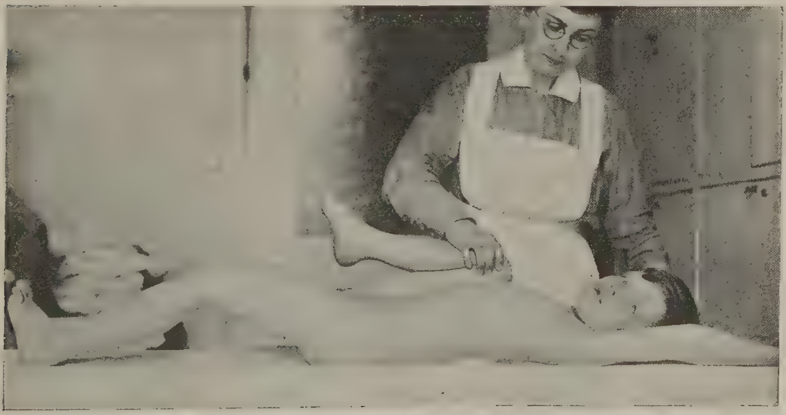

FIG. 113.—Determining the degree of flexion of the affected limb (left). The unaffected limb is flexed on the chest which fixes the pelvis and straightens the spine.

Sacro-iliac disease is a rare affection in children. It is differentiated from hip disease in that pain is referred to the abdomen, to the side of the pelvis, or to the thigh, and can be brought on by lateral pressure on the pelvis. Motion at the hip joint may appear to be restricted in all directions, but if the pelvis be steadied and the thigh slightly flexed to relieve the tension upon the psoas muscle, all the movements are normal with the exception of extension and inward rotation during extension; the weight in standing is borne on the well leg and there is frequently curvature of the spine, with convexity directed away from the affected side.

Congenital dislocation of the hip is characterized by a history of limping that has existed from the commencement of walking, unaccompanied by pain or other symptoms of disease. Upon examination there is shortening of the limb, usually not less than ½ in. when measured from the anterosuperior spine of the ilium to the inner malleolus, but no shortening when measured from the greater trochanter to the outer malleolus, which is explained by the trochanter being elevated above Nélaton's line. The head of the femur can be felt moving under the gluteal muscles on the dorsum of the ilium when the femur is rotated inward. Motion is restricted to a certain degree in extension, abduction and outward rotation. Roentgenograms demonstrate the dislocation.

Traumatic dislocation of the hip is the direct result of great violence and is sufficient to explain the immediate disability. The limb is held in the position of flexion, abduction and inward rotation. Shortening is present from the beginning, while in hip disease it occurs only in the later stages. The head of the femur can be felt moving under the gluteal muscles. A roentgen picture will assist in making a positive diagnosis.

Fracture of the femoral neck is not an uncommon accident, but is more liable to be of the green stick variety. It has markedly different symptoms from fracture in later life. The immediate results are much less disabling, and the child is frequently able to walk about within a few days after the injury. At this period it may be mistaken for hip disease. It is excluded (1) by the history—the patient going about prior to the time of injury without pain in the hip; (2) by the disability coming on immediately or shortly after the accident, and accompanied by pain only when the limb is moved; (3) by elevation of the trochanter with shortening of the limb; (4) finally, which is very important, by *x*-ray examination.

Coxa vara is an acquired deformity in which disease is absent. Movement is not limited in all directions as in hip disease, but only in abduction, flexion and inward rotation. Muscular spasm is absent, while shortening is present from the very onset with the greater trochanter elevated above Nelaton's line. The *x*-ray also furnishes conclusive evidence.

Prognosis.—Under favorable conditions tuberculous hip disease results in recovery in the majority of cases, but usually with more or less loss of function.

Functional Results.—In a certain number of cases perfect results may be obtained, but recovery with perfect motion is uncommon. Flexion deformity of less than 20° causes very little disability, while flexion beyond this amount increases the lumbar lordosis, making the buttock prominent (Fig. 117). Abduction, when of moderate degree, is an advantage, since it compensates for actual shortening of the limb. On the other hand, adduction, which gives an apparent shortening to the limb, is more disastrous than all the deformities, and should be prevented during treatment. Actual shortening due to arrested growth of the limb is beyond the control of the surgeon. Ankylosis of the hip, with the limb in extension and slight abduction, affects locomotion so little as to be scarcely noticeable.

Death may occur from pulmonary tuberculosis, tuberculous meningitis, amyloid degeneration of the viscera, from exhaustion and from intercurrent diseases. According to Calvé,* tuberculous meningitis occurs in multiple tuberculous bone lesions about four times as frequently as in the single.

Duration of Treatment.—An average case requires from three to four years of efficient treatment, followed by careful observation for at least another year.

Treatment.—In disease of the hip joint, as in other tuberculous joints, treatment to be effective should consist of complete rest and proper protection. The relief of symptoms occurs, as a rule, in those cases in which motion is eliminated, thereby preventing friction and its destructive effect.

The principles of rest and protection may be secured in a variety of ways, and must be considered from the standpoint of routine. But in the selection of treatment and in the general management of tuberculous hip disease, there are sometimes difficulties encountered, as well as complications, which may necessitate the modification of the routine treatment.

General hygienic measures, such as good nourishment, fresh air and sunlight are important, and have been described in the treatment of tuberculous spine disease.

Treatment by Recumbency.—Both in the early stage of the disease when there is slight flexion and abduction deformity, and in the progressive stage when the range of motion becomes more and more restricted; when the attitude changes to one of increased flexion and adduction; when pain is severe and constant; when abscess is present and when the constitutional effects of the disease are apparent, then recumbency with traction, by means of weight and pulley, should be employed.

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* Calvé, Prog. Med. (June 22, 1913).

The flexed leg should be elevated on an inclined plane, and traction made in the line of deformity (Figs. 114, 118). At the same time the leg should be placed in abduction. Five to eight pounds of traction force are usually sufficient for a child, but a working rule is that over the age of two, and up to five years, the weight or pull should be 1 lb. more than the age of the child in years. However, in each individual case close observation is necessary to determine the exact and most beneficial weight. It is best to begin with a weight of 2 or 3 lb., and then add as much more as can comfortably be borne by the child. Countertraction should be made by raising the foot of the bed.

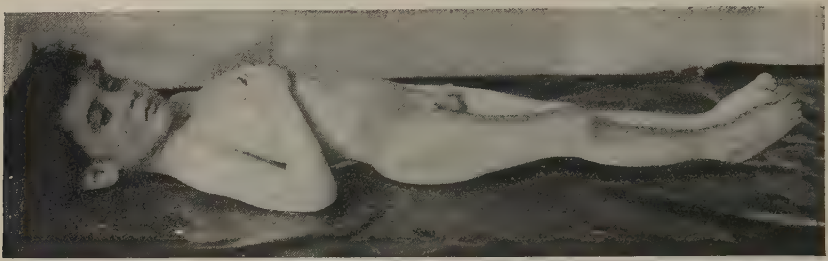

Fig. 114.—Instinctive effort at traction in hip disease by means of the toes of the unaffected limb.

Fig. 115.—A case of Perthes' disease which simulated hip disease (print retouched).

In order to assure efficient and constant traction, the patient must be prevented from sitting up. An efficient appliance for this purpose is the gas-pipe frame, covered with canvas, which has been described (p. 168). Upon this frame the patient can be fastened, the limb being elevated by a support which may or may not be attached to the frame.

Traction in bed nearly always affords prompt relief of the pain and slowly reduces the flexion deformity. This is shown in a few days, if a trial be made, that the angle of the inclined plane may be lessened, and the limb brought nearer to the bed, without provoking any spasm or lordosis. The weights are then re-applied, and as the mal-position of the limb diminishes the traction force is made gradually more in the long axis of the body (Fig. 118).

The duration of this form of treatment is difficult to estimate. Suffice to say it must be kept up for many weeks after the subsidence of the acute stage.

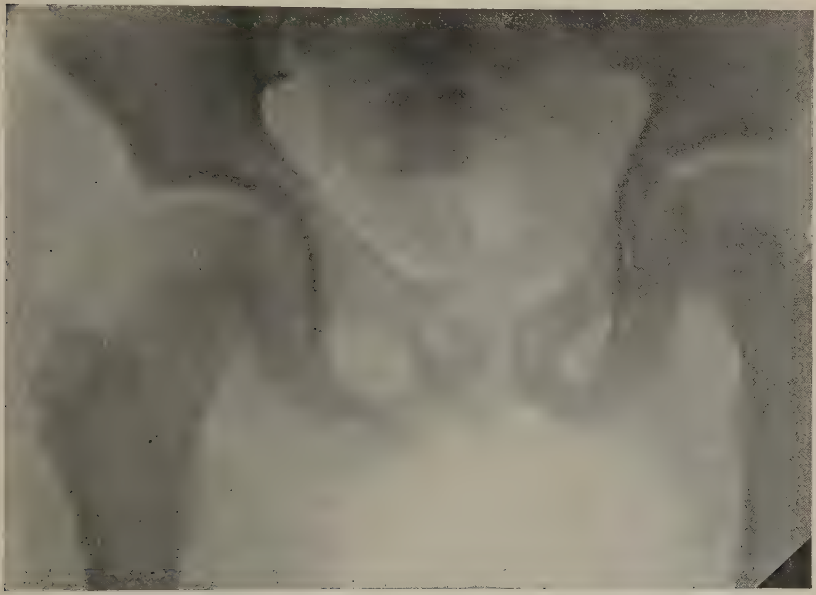

Fig. 116.—Tuberculosis of the ischium and pubis, symptoms of which simulated hip disease.

Treatment by Plaster-Paris Spica.—The plaster-paris spica is one of the most efficient and satisfactory methods of treatment, and may be carried out during the entire course of the disease. It has added advantages over other splints in that it is not difficult of application, the material is easily obtained and it is inexpensive.

In acute cases in which the joint is sensitive to movement, but in which deformity has been overcome by recumbency and traction, the limb may be put in a plaster spica and recumbency continued.

After all acute symptoms have subsided and have been quiescent for several weeks, without signs of abscess formation and the temperature under 100 degrees (Fahrenheit) the child may be allowed to go about, with the hip thoroughly immobilized. A long plaster spica extending well up on the trunk furnishes serviceable fixation. A high sole or patten is worn on the well foot, and locomotion is possible with crutches. Later the short plaster spica (Fig. 122) which permits motion at the knee, may replace the long plaster spica.

When the joint is no longer sensitive to movement, weight bearing may be tested by allowing the child to walk without the high sole. If no discomfort is experienced, then weight-bearing is continued. Finally, the crutches may be discarded.

In many cases of hip disease the symptoms are not acute, and the deformity present only in a slight degree. In such instances the plaster spica may be applied without preliminary recumbency. In the out-patient department of the Michael Reese Hospital, Chicago, a long plaster spica is applied to all tuberculous hip joints. The deformity in these patients is usually reduced by the repeated application of the spica. This obviates the necessity of hospital care, which otherwise would be required.

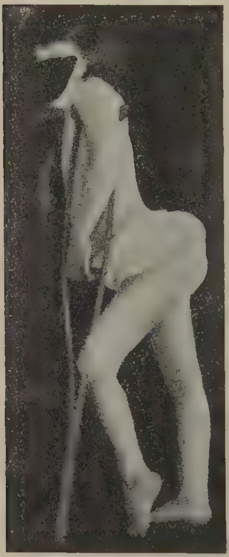

Fig. 117.—Deformity following a neglected case of hip disease. (Bradford.)

In some of the more resistant deformities, forcible reductions by manual traction under anesthesia is advisable. A long plaster spica is applied with the limb in extension, 20° abduction and 10° flexion and outward rotation. But forcible correction should not be employed, as pointed out by Whitman, "if deformity is of long standing,* and if the disease is active or of the destructive type, accompanied by infiltration of the tissue or by discharging sinus. Otherwise traction is to be preferred."

Application of Plaster-Paris Spica.—The plaster of Paris spica is usually made with the patient in the recumbent position, supported on a hip and shoulder rest (Fig. 119). An assistant supports the limbs and at the same time makes traction on the diseased one; a second assistant wrings the water from the plaster bandages, while a third one helps rub the different layers of bandages so that they may adhere to one another.

A seamless stockinette of sufficient length to cover the trunk and limb is cut in the shape of a one-legged drawers, sewed and then put on the child. Sheet-wadding is applied as a protection for the bony prominences, to prevent pressure sores (Fig. 123). An attempt should be made to correct any existing deformity. The plaster bandages should cover the lower half of the thorax and extend to the lower ends of the toes for a long spica, and to the knee for a short spica; it should be applied with the limb held at 20° abduction and with slight traction. The plaster splint is strengthened in front and back of the hip by carrying layers of the bandage up and down until an extra thickness has been added: Otherwise, the splint is liable to break at this point, because of the strain put on it. With a sharp knife the spica is trimmed about the groin and buttocks to prevent soiling from urine and feces. The ends of the stockinette are turned over the splint and sewed together.

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* Whitman, orthopedic surgery (1919), p. 349.

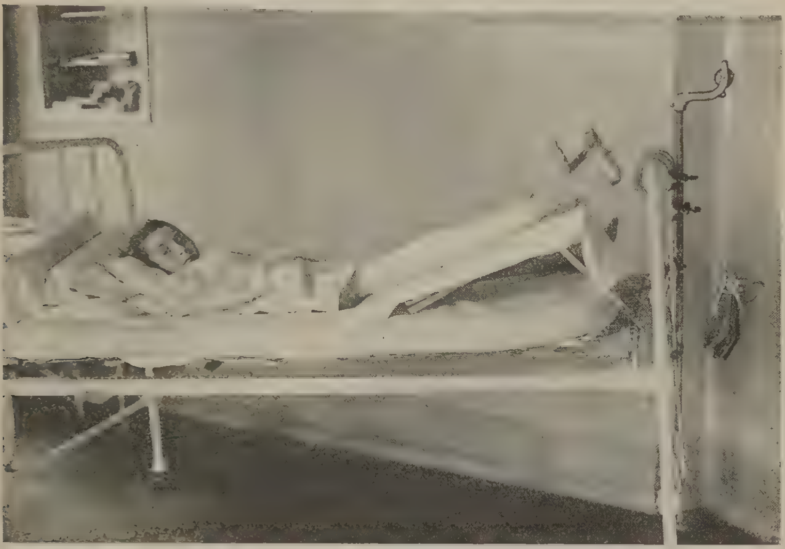

Fig. 118.—Traction in the line of deformity on inclined plane in hip disease.

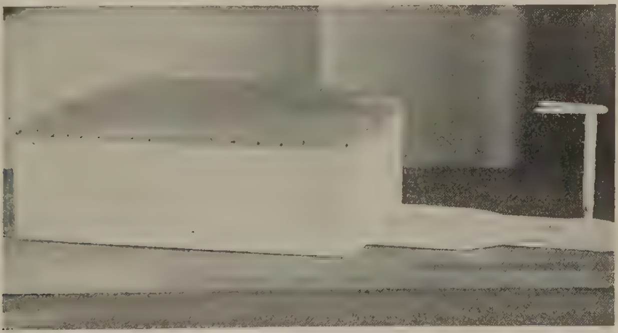

Fig. 119.—Support for the application of a plaster spica.

The splint need not be changed more often than every two months *if not uncomfortable and fairly clean.*

Mechanical Treatment.—Various forms of appliances have been devised for the adequate rest, protection and cure of the joint. Many of the claims originally made for these appliances have been abandoned, although in the supplementary treatment, they are, no doubt, of value. But the accurate fitting of a brace needs the services either of a mechanic or a trained orthopedic surgeon; then, too, careful and constant supervision is at all times necessary (Fig. 124).

The Bradford abduction splint is a most efficient mechanical splint. It consists of two steel rods, longer than the affected limb, connected below by a flat bar furnished with a small windlass attachment to furnish traction, and above by a ring open in front, obliquely placed upon the rods so as to fit the buttocks from the tuber ischii to above the greater trochanter, as in the well-known Thomas knee splint. Attached to the ring near the top of the inner rod is welded a bent steel rod, which passes above the symphysis pubis and under the perineum of the well side, and should be long enough so that the end should not press into the buttock when the patient is seated. The upper rings are padded and covered with leather, and if properly

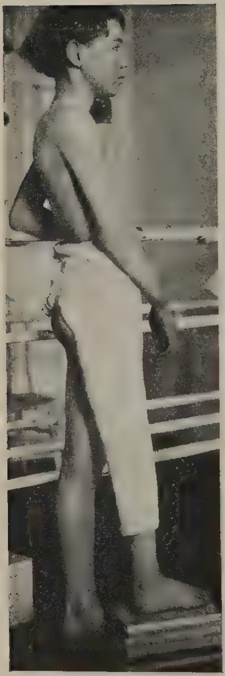

FIG. 120.—Plaster spica following excision of hip joint.

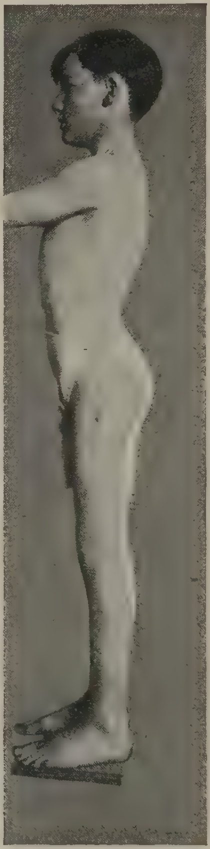

FIG. 121.—End result showing shortening in a cured case.

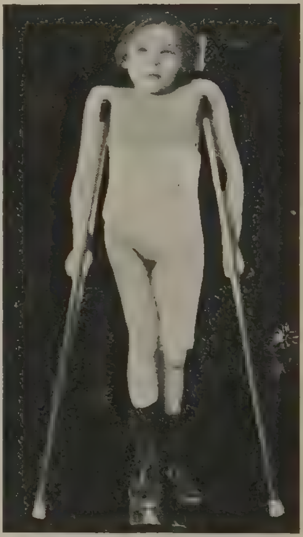

FIG. 122.—Short spica with a high sole on shoe on unaffected limb.

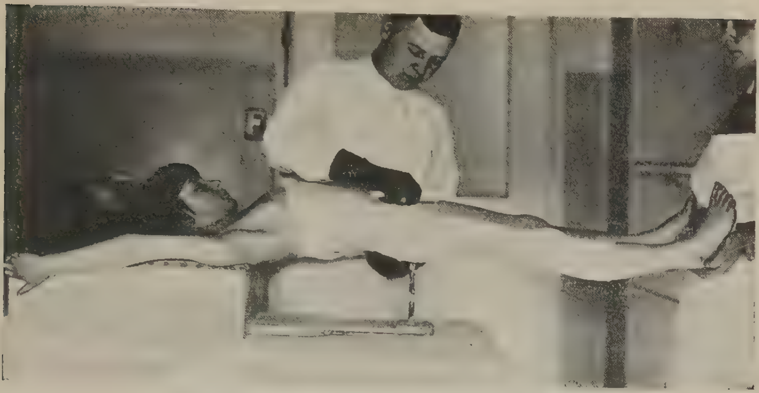

FIG. 123. –Application of a plaster spica.

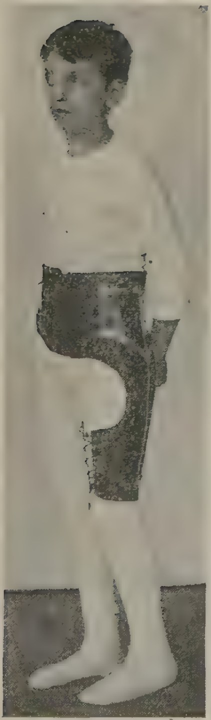

FIG. 124.—Convalescent hip splint.

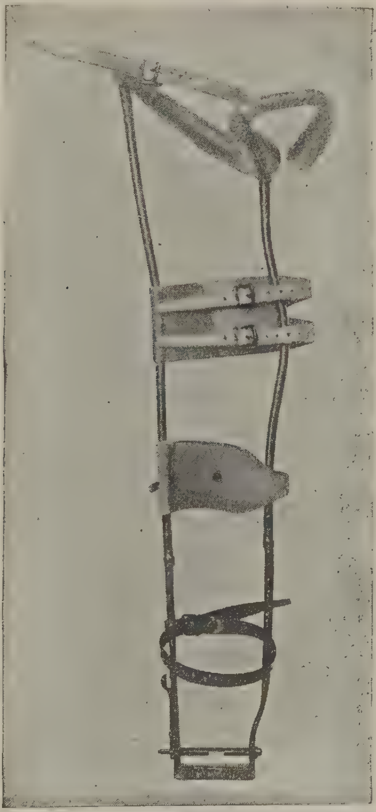

FIG. 125.—Bradford's abduction splint for traction in hip disease. (Bradford and Lovett, "Orthopedic Surgery.")

shaped are not soiled by feces or urine. Traction is furnished by means of adhesive plaster straps applied to the limb attached to webbing straps secured to the windlass. Circular leather straps steady the limb. Locomotion is made possible if a raised sole is applied to the foot of the unaffected side (Figs. 125, 126).

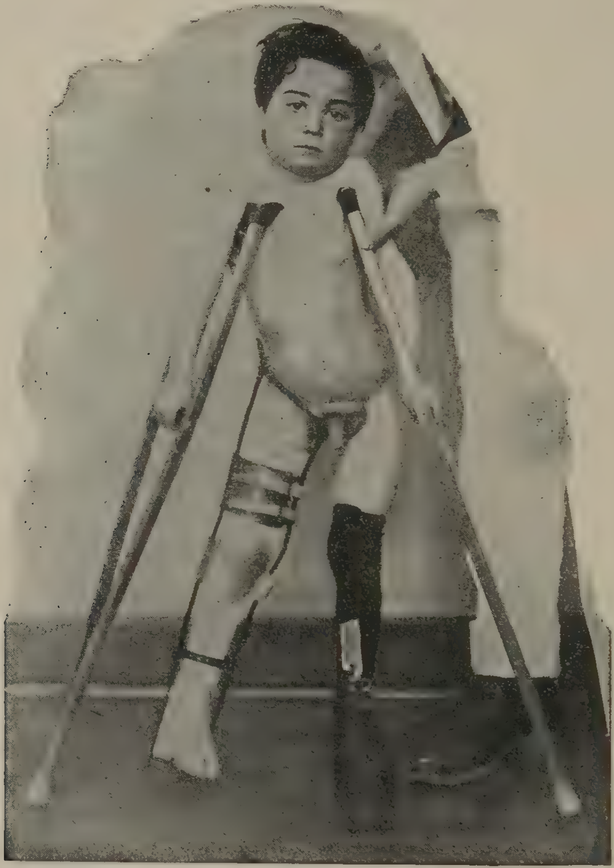

Fig. 126.—Abduction splint applied. (Bradford and Lovett, "Orthopedic Surgery.")

Whitman summarizes the treatment by traction splints as follows: "Under careful and constant supervision some traction may be exerted by an ambulatory splint, but under ordinary conditions the traction hip-brace is only efficient as a stilt in relieving the pressure and shock of weight-bearing. It does not prevent friction. The more accurate statistics of similar results in cases treated by this apparatus illustrate also its ineffectiveness in preventing deformity."

Thomas Hip Splint.—This splint is designed to immobilize the hip joint by an anteroposterior leverage. It consists of a steel bar passing down the back from the lower angle of the scapula to the lower third of the leg. It is bent to fit the back, thigh and leg. A band nearly encircles the chest below the arms, and bands also encircle the thigh and leg. The child can get about on crutches, aided by a high sole or patten on the well limb.

Operative Treatment.—Surgical procedure in tuberculous hip disease should be resorted to only as an *adjuvant measure* and not as the primary method of treatment in children.

As an adjuvant measure operative procedure is resorted to for correction of deformity; for the relief of pain in abscess formation due to distension of the capsule or tissues surrounding the joint; in extensive progressive, multiple tuberculous affections of the joints in which conservative treatment has been tried and failed, and in cases where symptoms of septic absorption are so severe as to endanger the life of the patient.

Correction of Deformity.—Under early and careful management serious deformity should not occur; however, if present in the acute stage of the disease, it should be corrected in the manner previously described (Fig. 121).

Flexion and adduction deformity with fibrous ankylosis may be reduced by manual force under anesthesia. But if there be continuous resistance to gentle manipulations, then it is useless to proceed. No attempt at the forcible reduction should be made for many months after the subsidence of acute symptoms. In quiescent or cured cases, division of the contracted adductor muscles and those attached to the anterosuperior spine generally is a preliminary procedure to forcible reduction.

If the deformity is fixed by bony ankylosis, no attempt at forcible reduction should be made, but osteotomy of the femur by Gant's or Adam's method should be performed. Following the operation the limb is placed in extension and abduction, and a long plaster spica applied, which should remain on for several months.

Abscess Formation.—Abscess formation, complicating joint tuberculosis, is best treated expectantly. If the abscess is left alone it disappears without treatment in at least 20 per cent. of the cases, and this percentage can be largely increased under the treatment by recumbency. A good rule to follow is not to interfere when the abscess remains quiescent and does not enlarge or cause symptoms. On the other hand, if it increases in size it should be aspirated, if necessary repeatedly, through a large needle. In a deep seated abscess within the joint of surrounding tissues, causing painful tension, incision is advisable under the most rigid aseptic precautions. The incision is immediately closed with sutures and sterile dressing firmly applied. The injection of iodoform and glycerine, 10 per cent., into abscess cavities has been frequently used. It is doubtful if it has any therapeutic value.

Treatment of Sinuses.—Tuberculous sinuses are best treated with cleanliness. The area surrounding the sinus should be daily washed in tincture of green soap and water, followed by the application of balsam of Peru on a sterile dressing.

Bismuth Paste.—It is claimed that bismuth paste injected into sinuses creates a leukocytosis. It is more reasonable, however, to consider the action of the paste as being chiefly mechanical, in that it plugs up the sinuses, dams back the pus, excludes the air and prevents entrance of pyogenic bacteria. These are favorable factors for the filling in of the sinuses with granulations.

Experience at the Home for Destitute Crippled Children, Chicago, has shown that bismuth paste injected into old sinuses of tuberculous joint disease may effect cure in a comparatively short period of time, but that new channels will form as an outlet for the tuberculous debris coming from the seat of the disease. This led to the abandonment of the use of bismuth paste.

Excision of the Hip Joint.—In children, the removal of the femoral head and neck not only involves a sacrifice of bone, and leaves a poor joint for weight-bearing, but it causes a crippling deformity in after years by the disturbance of growth of the bone; then, too, it is often but a partial operation, as frequently the acetabulum is primarily diseased and it is difficult and dangerous to remove. Only as a life saving measure is excision advisable. However, in early cases in adults, excision is often the operation of choice, depending upon the social and economic conditions (Fig. 120).

Multiple Bone Tuberculosis.—Calve* has made a valuable statistical study of the occurrence of multiple tuberculous bone lesions in children. In various groups of cases collected from the records of the Hospital Maritime at Berck, the multiple lesions have varied from 9.4 to 13. In 1910, at Berck, there were 85 cases of multiple foci, among a total of 869 cases. Of these, 27 were of the hip, 14 being double coxitis, and in nine the hip and spine were involved.

TUBERCULOUS DISEASE OF THE KNEE JOINT

Tuberculous disease of the knee joint ranks third in frequency of tuberculous joint involvement. The knee is one of the most accessible joints for examination, making early recognition of disease possible, and readily permitting splinting and control. Tuberculous knee-joint disease is synonomous with *white swelling and tumor albus.*

Etiology.—Tuberculosis of the knee joint is essentially a disease of childhood, the greater incidence of the disease being between the ages of two and three years. In adults it is relatively much more frequent than diseases of the spine or hip. Either sex is subject to attack without predilection. According to some authorities, the acute infectious diseases of childhood should be considered as important predisposing factors. The etiology in general has been discussed in the section on Tuberculous Spine Disease.

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* Calve, Prog. Med. (June 22, 1913).

Pathology.—The focus of disease may begin in the lower end of the femur, particularly in the region of the internal condyle, or in the head of the tibia, or occasionally in the fibula; rarely in the patella. It may originate, primarily, in the synovial membrane, and occurs relatively more frequent in the knee joint than in other joints.

The records of the Children's Hospital, Boston,* show that of 49 cases of knee-joint diseases, which were opened and examined at operation, showed a tuberculous bone focus, six involved all the bones of the joint, 18 both femur and tibia, nine the femur alone, five, the tibia alone, and one not classified. Nineteen showed actual bony destruction, three involving all the bones, nine the femur and tibia, 25 were noted as having synovial disease, though probably a much larger number was actually involved.

Nichols† believes that it is not possible to establish the origin of the infection of primary osteal disease unless the bones are sawn into sections.

Pathological conclusions as to primary osteal or synovial disease based on observation at the operating table, are of no particular value. From the clinical standpoint, however, two distinct types of tuberculous disease are recognized; a chronic synovitis, a type more often seen in adults; and an osteal type which seems to predominate in children.

In the osteal form of disease either primarily or secondarily involved, the same morbid process is seen as in other phases of bone tuberculosis. Accompanying the pathological changes in the bone there is a marked congestion of the synovial membrane with effusion into the joint. Fibrin is deposited upon the membrane, whichbecomes infected by the tuberculous process, and in turn invades the adjacent normal synovial membrane. The tuberculous granulation tissue gradually extends over the articular cartilage which finally becomes infected by the diseeas. In the meantime, the synovial membrane and capsule have become thickened, and this gives a peculiar elastic resistance on palpation. Seldom is there much fluid in the joint when thickening of the tissues has occurred.

Symptoms.—*Lameness.*—Limping, is the first symptom, generally occurring especially after fatigue. It subsides with rest, recurs on activity, finally becoming constant. This is due not only to the sensitiveness of the joint, but to the flexion deformity which usually accompanies it.

Pain.—This, as a rule, is not severe, except during the acute exacerbation, when it is brought on by any sudden twist or jar. "Night cries" are less common than in hip disease.

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* Sever and Fiske, Tuberculosis of the knee joint in childhood, American Journal of Orthopedic Surgery (April, 1915).

† Nichols, Trans. Amer. Orthopedic Assoc., xi.

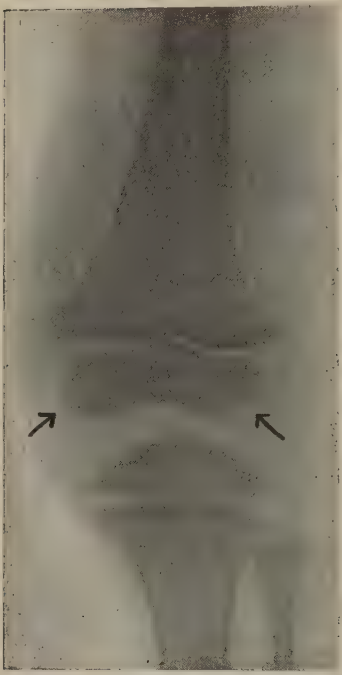

FIG. 127.—Tuberculous osteitis of the knee.

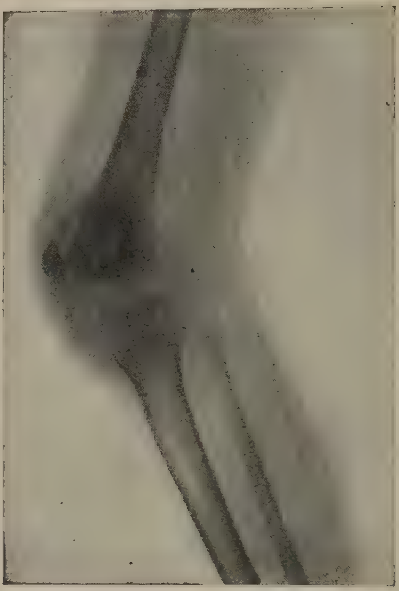

FIG. 128.—Tuberculous synovitis of knee with beginning flexion deformity.

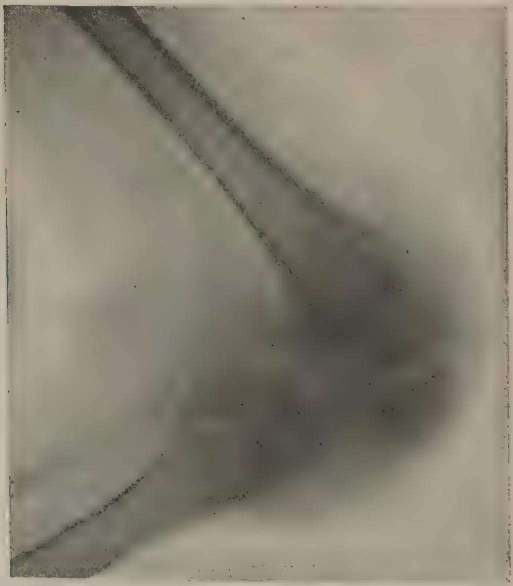

FIG. 129.—Neglected tuberculous knee showing flexion deformity and swelling.

Tenderness to pressure can usually be elicited over the inner and lower surface of the internal condyle of the femur.

Swelling.—The earliest evidence of swelling is absence of the depression on either side of the patella, due not only to an increased amount of fluid within the joint, but more especially to thickening of the peri-articular tissues. Much enlargement of the joint, with distinct fluctuation as a rule denotes abscess formation. In advanced cases the skin becomes reddened, and eventually the abscess opens spontaneously, if not aspirated or incised. The enlargement of the knee is more apparent than real, because of the muscular atrophy above and below the joint. In some cases the distention of the skin makes it look white and anemic, hence, the name "tumor albus."

Atrophy.—Atrophy of the muscles of the thigh and calf is present, depending upon the duration of the disease and interference with function.

Heat.—Local heat of the affected joint is present almost from the beginning of the disease. If persisting, it indicates continued activity of the diseased process which in turn, calls for more efficient treatment.

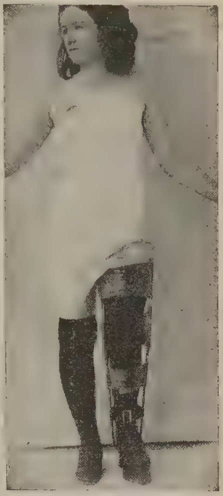

FIG. 130.—Retardation of growth of leg as a result of tuberculous knee disease. Caliper splint applied with high heel and sole.

Deformity.—Flexion deformity is characteristic of the disease; it occurs gradually and if not early corrected, subluxation of the head of the tibia may follow. The greater power of the flexor muscles of the thigh over the extensors explains the constant tendency to deformity (Figs. 127, 129).

Muscular Spasm.—The normal range of motion is limited by muscular spasm. Even in the early stage of the disease complete extension and flexion are not impossible. As the disease progresses, stiffness and flexion deformity become more marked.

Growth of Limb.—At times the tuberculous process in the epiphysis irritates the bone to increased growth—osteoplasia—and the affected leg becomes longer, from ¼ to 1 in., during the active stage. This increase in length involves the femur and often may be detected within six months of the onset of the disease. If the disease has been severe and of long duration, there is a retardation of growth of the tibia, and the final result is a shorter limb (Fig. 130).

Abscess—Abscess is a very frequent complication and may appear as a purulent distention of the capsule, or in the peri-articular tissues.

Differential Diagnosis.—Injury.—Traumatic synovitis, if persistent, can be distinguished from tuberculous knee disease only by continual and painstaking observation.

Infectious Arthritis.—The affection begins suddenly, with chills, high fever and other septic symptoms, accompanied by pain in the joint.

Arthritis Deformans.—This condition is uncommon in children. It usually is polyarticular.

Hysterical Joint.—A hysterical knee is unusual in young children. The subjective symptoms may be simulated, but the physical signs are absent.

Sarcoma.—In the early stage, sarcoma of the lower end of the femur may readily be mistaken for and not easily differentiated from tuberculous knee-joint disease. Its rapid growth may be the first to excite suspicion. A roentgenogram will usually show greater destruction of bone in sarcoma, while in tuberculosis there is usually involvement of the joint.

Syphilis.—Hereditary syphilis of joints in children is usually secondary to neighboring bones.

Hemophilia.—Very often this condition resembles tuberculous knee-joint disease. The rapid onset, the history of the bleeder's diathesis, and the absence of inflammatory symptoms should make differentiation comparatively easy.

Prognosis.—When treated early and efficiently the result obtained in tuberculous knee-joint disease is not only better as to function and deformity, but the duration is shorter than in spine or hip disease. Perfect function is sometimes restored, but, as a rule, recovery takes place with more or less limited motion, some deformity, yet still a serviceable leg.

The following details of 150 cases of knee-joint disease treated at the New York Orthopedic Hospital* are of interest.

Average age on admission, years	6½
Average duration of disease on admission, years	2
Average duration of treatment, years	7
Number of patients cured	106
Number of patients under treatment at present time	7
Number died	7
Number could not be located	43
Of the cases cured, number with lengthening of limb	10
Number with no change in the length of the limb	73
Number with less than 1 in. shortening	7
Number with less than 2 in. shortening	14
Number with more than 2 in. shortening	2
Number with flexion deformity without ankylosis	5
Number with flexion deformity with ankylosis	13
Number with ankylosis without deformity	13

In private practice the duration of treatment is two to three years. Relapses are of frequent occurrence.

Treatment.—The principles of treatment in tuberculous knee-joint diseases are, as in other tuberculous joints, complete fixation and protection. Fixation of the joint is accomplished by means of either a plaster-paris cast or a brace, such as the Thomas knee splint (Fig. 131).

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* Humphries and Durham, Journal of American Medical Association (Jan. 27, 1917).

Treatment by Plaster-Paris Cast.—When the joint is fixed by a plaster cast it should extend at least from the groin to the ankle. A common error is to put on too short a cast. The cast is applied with the child in the recumbent position. An assistant grasps the ankle and makes traction on the limb to overcome muscular rigidity. In this way a certain amount of deformity can be overcome. The limb is protected by sheet-wadding, which should be thicker over the knee joint to prevent pressure sores. A bandage is firmly wrapped around the wadding, and then plaster bandages are smoothly applied.

If the disease is particularly active, as shown by severe pain, much muscular spasm and flexion deformity, a plaster cast should be applied, which includes the foot and pelvis; then, too, confinement to bed is essential.

After the acute symptoms have subsided, or in the very early stages of the disease, in which no acute symptoms are present, the child may be allowed to walk, but the affected limb must be protected from the jar of weight-bearing. This is done by means of crutches and by a high sole or patten on the well leg, which lifts the diseased limb from the ground.

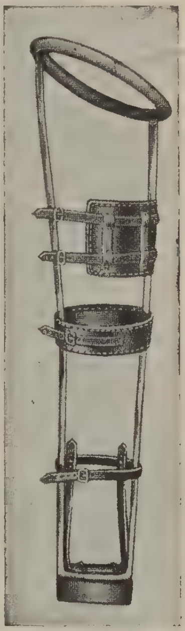
FIG. 131.—Thomas knee brace.

Mechanical Treatment.—The Thomas brace for tuberculous knee-joint disease is an excellent immobilizing apparatus, which, at the same time, protects the limb from weight-bearing. Two forms are in general use—the caliper and knee splint.

The caliper splint (Fig. 131) consists of a ring and two side bars of strong iron wire. The ring slopes from without inward and from before backward, so that the part upon which the tuberosity of the ischium rests is its lowest part; it is bent to such a shape that when properly padded it shall fit the outline of the upper thigh, following fairly accurately the gluteo femoral crease and the groin. The ring is padded with felt and covered with basil leather. To the side bars, two strips of leather, 2 and 3 in. in width, are sewn to form a support for the back of the ankle and knee when the splint is applied. The lower ends of the bars are bent inward, at a right angle, to be inserted into a steel tube in the heel of the shoe. The splint should be sufficiently long so that the child's heel, in walking, is elevated a full inch from the inside of the shoe. It is applied by slipping the ring on over the leg and pushing it well up against the tuberosity of the ischium; the bent ends of the side bar are then inserted into the hole in the heel. The limb is held extended by pressure pads applied, in front, to the thigh and leg. Deformity, if present, is reduced gradually by the direct pressure of the pads.

The writer has modified the caliper splint to allow lengthening, according to the growth of the child, and also to overcome flexion deformity (Fig. 132).

The Thomas knee splint (Fig. 131) is similar to the caliper splint except that it terminates 2 to 3 in. below the foot, and takes the weight on a cross-bar padded with leather or rubber. It is worn with a 2 to 3-in. sole on the shoe of the unaffected side. A strap is attached to each side of the cross bar to provide for traction on the limb, as well as to hold the brace in place. The splint is buckled to adhesive plasters, which extend from the knee to the ankle.

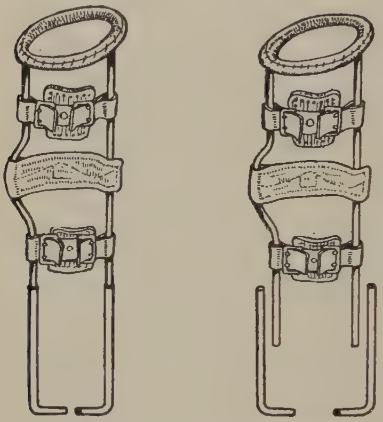

FIG. 132.—Jacobs' modified knee splint. As may be noted from the illustration each of the lateral uprights consist of two pieces. The upper piece is inserted into the lower one where lugs of different sizes allow lengthening at will.

Treatment of Deformity.—Flexion deformity occurring during the early stage of disease does not form an insurmountable obstacle to the restoration of the joint to a proper position. The deformity may gradually be overcome by means of repeated applications of the plaster cast, together with manual traction or by the Thomas or Jacobs modified caliper splint. Rapid reduction of the deformity may be performed by careful manual traction under anesthesia; care being taken to pull the head of the tibia forward. A plaster cast is then applied.

If the deformity is of long standing the obstacle to reduction is caused not only by muscular rigidity, but by structural shortening of the ham-string muscles. In such cases the danger of forcible correction may result in the increased tendency to backward and outward dislocation of the tibia. For this reason the shortened tendons are divided, under anesthesia, traction being made by an assistant, while the surgeon endeavors to straighten the knee by pushing the condyles of the femur backward with one hand, and with the other, supporting from behind, he pushes upward. After correction a plaster cast is applied.

Flexion deformity with bony ankylosis may be corrected by linear or cuneiform osteotomy above the knee.

The Treatment of Abscess is similar to that of tuberculous abscess elsewhere.

Radical Surgical Treatment.—*Excision of Knee Joint.*—Excision for tuberculous arthritis has the greatest application and the greatest

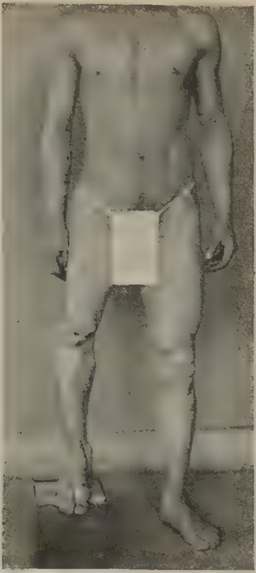

FIG. 133.—Case of tuberculosis of the knee which was resected ten years previously. Note the amount of shortening during the interval.

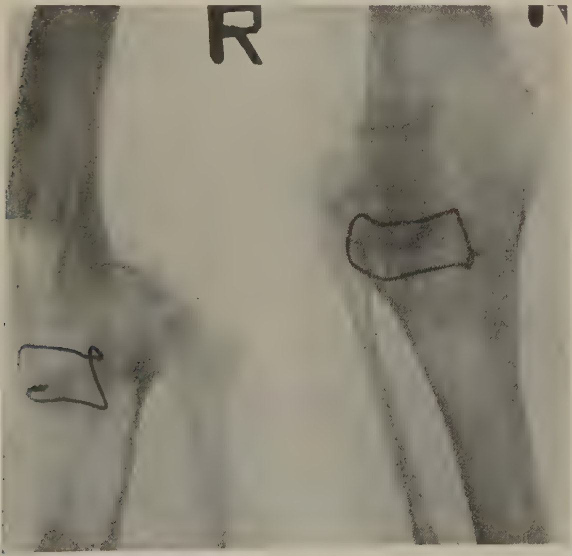

FIG. 134.—*X*-ray of case shown in Fig. 133, showing bony ankylosis.

probability of success in adults, inasmuch as it shortens the period of disability with a presumption of a rapid cure. In children excision of the knee joint does not accomplish desirable results. Removal of the epiphysis of the femur and tibia results in defective growth of the bone which explains the marked shortening of the limb encountered in later life (Figs. 133, 134). Excision in the child is undertaken in exceptional cases only.

Amputation.—In extreme cases of disease of the knee joint, when the limb is riddled with discharging sinuses, and symptoms of septic absorption are so severe as to endanger the life of the child, amputation at the thigh is of vital indication.

TUBERCULOUS DISEASE OF THE ANKLE AND TARSAL BONES

Tuberculous ankle disease ranks next in frequency to tuberculous knee-joint disease, and fourth with other tuberculous joint diseases. Tuberculosis of the tarsal bones is of less frequent occurrence than ankle disease.

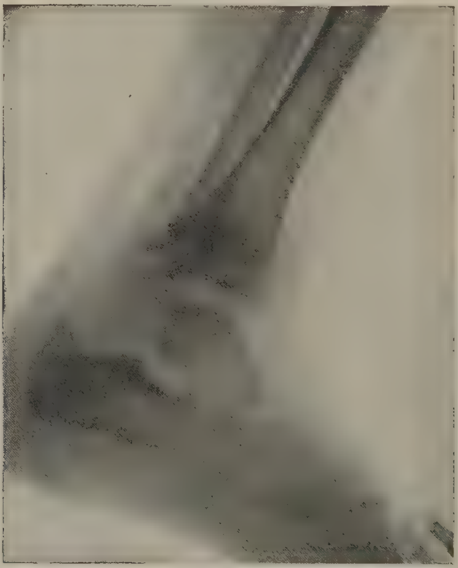

Fig. 135.—Tuberculous disease of the ankle showing involvement of the os calcis and fibula.

Etiology.—The etiological factors in disease of the ankle and tarsal bones are in no way different from other tuberculous joints.

Pathology.—It is a matter of controversy as to whether the disease begins more frequently in the synovial membrane or in the osteal tissue. Of the bones entering into the formation of the ankle joint, the focus of the disease is more often in the astragalus, next in frequency in the tibia,

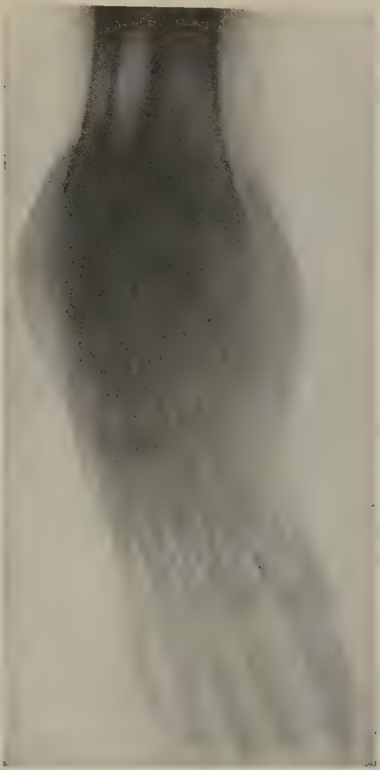

Fig. 136.—Tuberculous synovitis of the ankle.

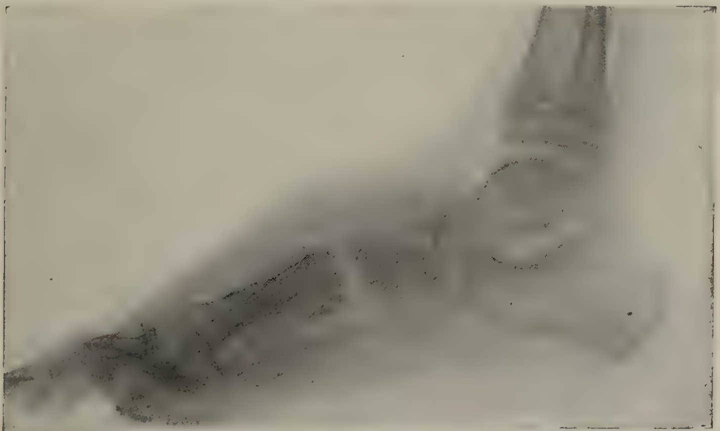

Fig. 137.—Tuberculosis of the mid tarsal bones.

and least often in the fibula. Of the tarsal bones the calcaneum is the one most frequently involved (Figs. 135–137).

Hahn has tabulated 1231 cases of tuberculosis of the ankle and foot, with reference to the location of the disease, and found that 26 per cent. occurred in the calcaneum; 24 per cent. in the astragalus, 13 per cent. in the cuboid, 9 per cent. each in the scaphoid, cuneiform and metatarsals, 8 per cent. in the malleoli, and 2 per cent. in the phalanges.

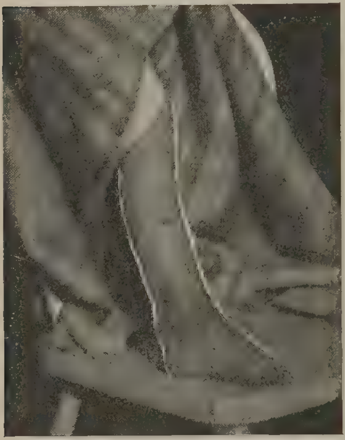

Fig. 138.—Old tuberculous ankle showing sinuses and swelling.

Symptoms.—The onset of tuberculous disease in the ankle or foot is insidious. The earliest symptom is *lameness*, which, at the beginning is noticed only at the end of the day. This finally becomes constant and permanent. The child unconsciously attempts to relieve the foot from weight-bearing and from movement. *Pain* may not be a prominent symptom during the early stage; later it may be continuous, worse at night or upon sudden movement or jar. *Sensitiveness* to pressure is usually elicited over the site of the infection. *Swelling* is due to infiltration of the tissues and varies with the site of the disease. In the ankle the swelling appears first about one or both malleoli. If the calcaneo-astragaloid joint alone is affected, the swelling lies chiefly below the malleoli; if the tibiotarsal joint is simultaneously involved, swelling is in front of the ankle (Fig. 138). In advanced cases the swelling over the foot, between the mediotarsal and tarsometatarsal joints, spreads rapidly so that it is not possible to determine the primary focus of the disease *Deformity* of the foot varies, depending upon the location of the disease. If the focus of infection is in the ankle joint, the foot usually assumes the position of equinus; if it is in the calcaneo-astragaloid joint, a position of eversion is generally assumed and if the astragaloscaphoid joint is the one affected, a position of persistent abduction is assumed. *Motion* is restricted as the result of muscular spasm. Restricted anteroposterior motion usually is found in ankle disease, while restricted lateral motion is more often found in tarsal disease. *Atrophy* of the muscles of the leg occurs early, increases as the disease progresses, and is greater than that following disuse alone.

Abscess occurs in about 75 to 85 per cent. of tuberculous ankle disease; it most frequently points on the inner side or dorsum of the foot—rarely on the plantar surface.

Differential Diagnosis.—In **sprain** there is generally obtained a history of injury; the symptoms are acute and motion is not limited as in ankle disease. In **flat foot** the discomfort is referred to the mediotarsal joint, and is of a strained and aching feeling after use, which rapidly disappears on complete rest; sensitiveness to pressure is uncommon. The discomfort in ankle disease is almost always unilateral, in contrast to flat foot, which is bilateral. Then, too, roentgenograms show no pathology in the latter. The swelling and tenderness of **tenosynovitis** are localized along the course of the tendons, and crepitation can be felt over the tendons on voluntary movement. Pain occurs only on motion, and there is no muscular spasm or deformity.

Prognosis.—If treated early, the final result is very good in children. Considerable motion at the ankle joint is often retained.

In Humphries and Durham's series of 23 cured* cases of ankle-joint disease, 15 had free motion with no deformity of the foot; 6 had limited motion with no deformity, and 2 had limited motion and deformity of the foot.

Treatment.—The principles of treatment to be followed are the same as in other tuberculous joints. Fixation of the part can be accomplished best by a plaster-Paris cast, extending from the end of the toes to the knee joint; the foot should be at right angles, to the leg and slightly inverted. In the early acute cases with much swelling and pain on movement, the child should be placed in bed. After the subsidence of the acute symptoms, or in early cases without acute symptoms, the child may be allowed to be up and about, with crutches and a high sole on the shoe of the normal foot. Later, weight-bearing is permissible. If deformity is present, it should be gradually corrected at each application of the plaster cast, until the desired correction is attained.

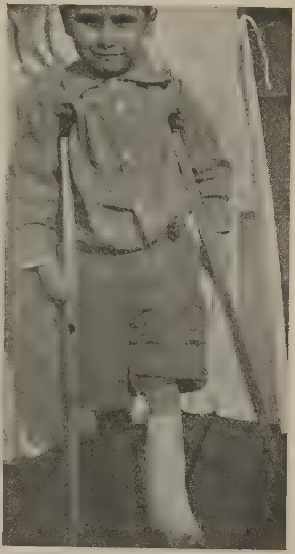

FIG. 139.—A tuberculous ankle in a cast.

Abscesses are to be treated like those occurring in tuberculous disease elsewhere.

Operative Treatment.—In no disease are surgical measures more justified than in tuberculous disease of the ankle or foot. They will shorten the long period of treatment necessitated by conservative methods. But it takes courage to advise operation in children, in the early stage of the disease, as there is a probability of cure by conservative methods.

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* Humphries and Durham, Journal of American Medical Association (January, 1917).

If the tuberculous disease is confined to the astragalus, astragalectomy is indicated. If the morbid process is localized in any of the tarsal or metatarsal bones, then removal of the involved bone is necessary. If the disease is limited to a single focus in the os-calcis, it is removed by chiseling; but if the disease is at all extensive, then the entire bone should be removed.

In some cases, the tuberculous disease is too extensive to permit radical removal; in such cases, fixation and improved hygienic surroundings are indicated.

In bony ankylosis of the joint with the foot in the position of valgus or equinus valgus, motion can be restored and the deformity overcome by the removal of the astragalus.

TUBERCULOSIS OF THE SHOULDER JOINT

Tuberculous disease of the shoulder is an infrequent disease of childhood. Of 7474 cases of bone and joint tuberculosis, treated at the Children's Hospital, Boston,* from 1869 to 1909, there occurred 17 cases of tuberculosis of the shoulder.

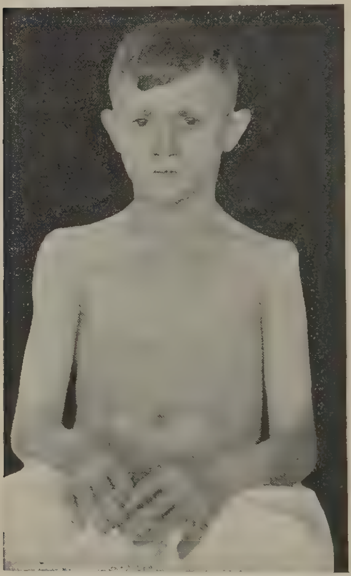

FIG. 140.—Muscular atrophy in tuberculous disease of the shoulder.

Pathology.—The disease usually commences as an osteitis; rarely as a synovitis. The head of the humerus is commonly affected: seldom the glenoid fossa. Two types of caries are to be distinguished. In one type the progress of the disease is rapid, abscess forms and appears near the insertion of the deltoid muscle; in the other type, known as "caries sicca," the progress of the disease is slow, with no swelling nor abscess. Nevertheless, the morbid process, in some cases, is so great that it destroys not only the head of the humerus, but the shaft as well.

Symptoms.—The disease is insidious in onset. *Pain* is usually the first symptom, worse at night, being of a dull aching character, referred to the joint. *Sensitiveness* to pressure is elicited in the neighborhood of the coracoid process. It is not unusual for the patient to complain of the inability to lie on the affected shoulder. *Limitation of motion* is characteristic. The child is unable to abduct, elevate or rotate the arm inward or outward without the scapula moving the humerus. This inability to extreme movement is the result of muscular spasm. *Swelling* of the shoulder joint occurs early, but soon afterward there is a gradual flattening of the joint, due to *atrophy* of the deltoid muscle, or, as in the caries sicca type of the disease, to the progressive destruction of the head of the humerus (Fig. 140).

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* Sever and Fisher, Amer. Journal Orth. Surg. (April, 1915).

Abscess is denoted by swelling and infiltration of the tissues about the shoulder, increased local and general temperature, and finally by burrowing of pus.

Prognosis.—As the disease is very uncommon in childhood, statistics are of little value relative to the end results of this affection. One may expect the caries sicca type to heal in two to three years with fibrous ankylosis or with destruction of the head of the humerus. Arrested growth of the arm may become evident later.

Treatment.—To assure fixation of the joint, the forearm is flexed at an acute angle and held by a sling passed around the neck and wrist: the arm is held to the side by a bandage, or, what is better, an adhesive-plaster dressing. But a plaster-Paris spica with the arm held in abduction forms a more permanent dressing and assures continuous rest.

Operative Treatment.—*Excision* of the joint may be often advisable in adults, but it should not be undertaken in children. As the upper epiphysis contributes mostly to the growth of bone in length, its removal causes a disturbance of growth of the limb. *Arthrectomy* is preferable when the removal of the focus of the disease is to be considered.

TUBERCULOSIS OF THE ELBOW JOINT

Tuberculous disease of the elbow is fifth in order of frequency, as compared with other tuberculous joints. Of 59 cases of disease of the elbow joint reported by Whitman* 45 were under 11 years of age.

Pathology.—The focus of disease may begin primarily in the synovial membrane, but more commonly is found in the osseous tissue. Of the bones entering into the elbow joint, the olecranon process of the ulna is the most frequent site; then the external condyle of the humerus; and only rarely does the focus begin primarily in the head of the radius (Fig. 141).

The pathological changes occurring in the joint are similar to those of other tuberculous joints.

Symptoms.—The two earliest symptoms of tuberculous elbow disease are *limitation of motion* and *swelling*. *Limitation of motion* first makes its appearance in the form of *stiffness;* gradually there is resistance to complete flexion and extension as well as to pronation and supination—the result of muscular spasm. Eventually, the joint becomes fixed, with the forearm in the position midway between flexion and extension. This *deformity* is quite characteristic of the disease. *Swelling* is at first slight, merely obliterating the depression on either side of the olecranon process; later it becomes more general, due to thickening of the tissues. The joint now assumes the characteristic fusiform shape (Fig. 142). *Pain* may be an early symptom. It is referred to the elbow joint and increases with movement. *Sensitiveness* to pressure varies in degree and is elicited at the site of the disease. Muscular *atrophy* appears early and varies with the functional disability of the joint.

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* Whitman, orthopedic surgery (1919), p. 455.

FIG. 141.—Tuberculous disease of the elbow with later proliferation.

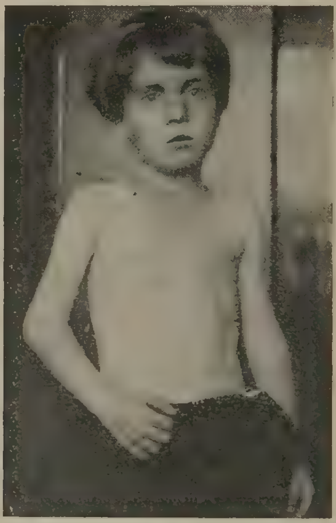

FIG. 142.—Characteristic position and fusiform swelling in tuberculous elbow disease.

Abscess is of frequent occurrence and usually opens just below the condyles. If the destructive process is very extensive, the soft parts are riddled with sinuses.

Prognosis.—The prognosis as to life is good. As to function, a fair range of motion is possible though shortening may be present. The average duration of treatment is about two and one-half years.

Treatment.—The joint should be fixed at a right angle with the forearm in a position midway between pronation and supination. This is the most advantageous position with regard to function if the disease terminates in ankylosis. A plaster-Paris cast is far superior to any other fixation support, and should extend from the hand to the axilla.

Reduction of Deformity.—If the joint is held stiff with the forearm in an attitude of semi-extension, it may be corrected gradually by bringing the forearm into as much of a flexed position as possible, and applying a plaster cast. The application of each cast secures an improved position of the limb and eventually the deformity is corrected.

Deformity may be corrected by a sling passed around the neck and wrist. The forearm is flexed as much as the muscular fixation will allow, and the head carried forward (Fig. 143). In a few days the head will be erect and the forearm will be in a changed position (Fig. 144).

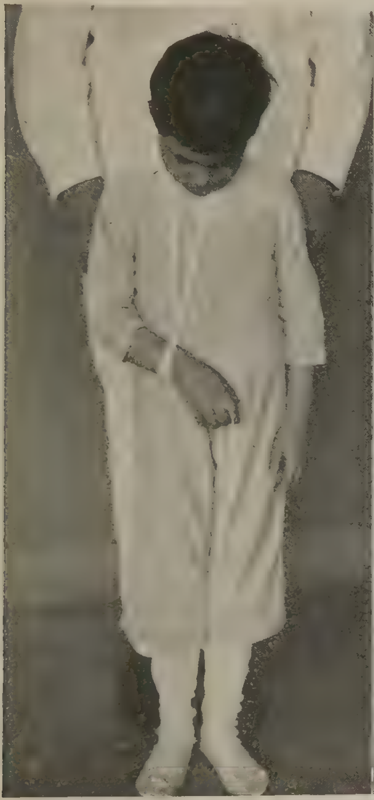

Fig. 143.—Correcting the faulty attitude in tuberculous disease of the elbow joint by flexing head and attaching sling to neck and wrist.

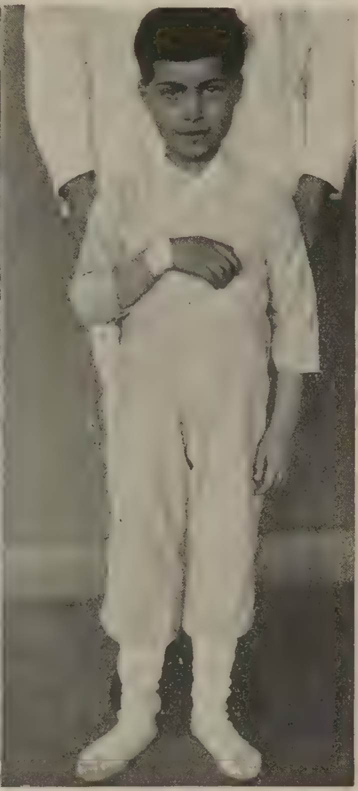

Fig. 144.—The faulty attitude corrected by successive stages.

The same procedure is repeated until the desired degree of flexion of the forearm is obtained. In some instances, it may be necessary to give an anesthetic to correct the faulty attitude. The joint is then immobilized by a plaster cast.

In bony ankylosis virtually a new joint may be formed by means of an arthroplastic insertion of flaps of fascia between the created bone surfaces, but such operation should not be undertaken until several years after the disease has been apparently cured.

Abscess.—The treatment of abscess of the elbow is in no way different from that of tuberculous abscess elsewhere.

TUBERCULOSIS OF THE WRIST JOINT

Tuberculosis of the wrist in childhood is relatively infrequent compared to ankle joint disease.

Pathology.—The pathological changes in the wrist joint are similar to those found in other tuberculous joints. The focus of disease may occur primarily in the bones or synovial membrane and it is a mooted question as to which is the more common. On account of the extensive arrangement of the synovial membrane, all parts become finally involved. When the disease commences in the bone, the primary focus may be in the radius, carpal bones or metacarpals. The tendon sheaths may become affected secondarily.

Symptoms.—The earliest manifestations of the disease are *pain* on motion, *stiffness* and *swelling*. As the disease progresses stiffness increases until there is virtually no movement at the wrist joint as the result of *muscular spasm*. *Swelling*, too, increases, so that the bony outlines of the wrist are no longer manifest. *Atrophy* of the muscles of the forearm occur. And the wrist now has the characteristic fusiform shape. *Sensitiveness* to pressure is most marked over the seat of the disease and the temperature of the joint is elevated. *Flexion deformity* of the hand with the fingers straight and the thumb extended is not uncommon. *Abscess* formation is very common and eventually opens on the dorsum of the wrist or at the side of the flexor carpi radialis tendon.

Prognosis.—In children the prognosis is usually good as to function. The ultimate result as to motion depends, of course, upon the extent of the disease and the effectiveness of mechanical treatment.

Treatment.—The treatment consists of fixation of the joint, which can be most effectively carried out by either a light plaster-Paris cast, extending from the elbow to the first phalangeal joints, or a molded leather splint. The hand should be maintained in an attitude of dorsal flexion so that the fingers will be able to close more easily if the wrist becomes ankylosed. Any flexion deformity may be easily overcome by the application of plaster cast every two or three weeks. The treatment of abscess is the same as in other tuberculous joints.

Surgical Treatment.—In children tuberculous disease limited to a single bone of the carpus should be first treated conservatively, but if the disease becomes extensive, then removal of all the carpals is indicated. Excision is also indicated in flexion deformity of the wrist with bony ankylosis.

TUBERCULOUS DACTYLITIS (SPINA VENTOSA)

Tuberculous disease of the long bones of the hand and foot attacks children before the fifth year of age, most frequently in the second and third years.

Pathology.—The infection may be central, *i.e.*, in the diaphysis; rarely is it subperiosteal. The osseous tissue is gradually destroyed while at the same time new bone, not distinguishable from the old, is being formed under the periosteum, so that the shaft becomes very much increased in circumference, and spindle in shape; the cavity is filled with boggy granulation tissue and fragments of sequestrated bone (Fig. 145). The joints themselves remain long intact, but may be invaded later by perforation through the epiphysis. The disease is more frequent in the hand than in the foot; the metacarpal bones and the phalanges are affected about equally, while in the foot, the metatarsals, particularly the first, are more often attacked than the phalanges of the toes. The disease is not infrequently found simultaneously in several phalanges or metacarpals of the same hand (Fig. 146). It is also not unusual to find spina ventosa associated with tuberculous lesions in other parts of the body.

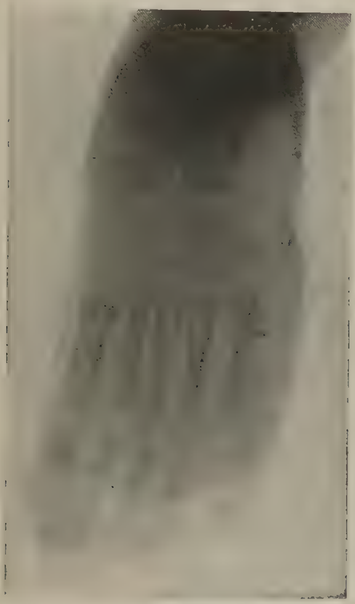

FIG. 145.—Tuberculous dactylitis involving the 5th metatarsal.

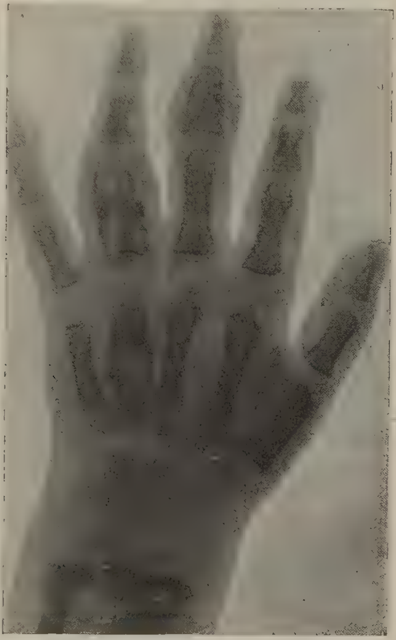

FIG. 146.—Tuberculous dactylitis of the second phalanx of the middle finger, first phalanx of ring finger and fourth metacarpal bone. (Steindler.)

Symptoms.—The onset is insidious. The earliest symptoms are *swelling* (Fig. 147) and *tenderness* to *pressure*. The swelling, at first localized, gradually becomes diffused. At this stage the disease may be arrested and repair take place under treatment. But more often the disease progresses, the bone is partly destroyed, and the neighboring joint invaded.

Differential Diagnosis.—Tuberculous dactylitis may be confused with hereditary syphilis, from which it is very often difficult to differentiate. Calot ("Indispensable Orthopedics" 11, 6th ed.) states that one-third of all cases of spina ventosa are syphilitic, one-third mixed, that is syphilitic and tuberculous, and one-third purely tuberculous. In syphilitic dactylitis there is diffuse uniform thickening of the phalanges; the skin is usually brownish or copper in color and almost painless. Roentgenograms shows periostitis with new bone of increased density, symmetrically deposited on both sides of the shaft, which is easily distinguishable from the old. A Wassermann test aids in the diagnosis. But in doubtful cases Calot institutes a test treatment with mercury or iodide of potassium, insisting more particularly on the *iodide*. In *osteosarcoma* the progress is rapid, the tumor soft, and of variable consistency.

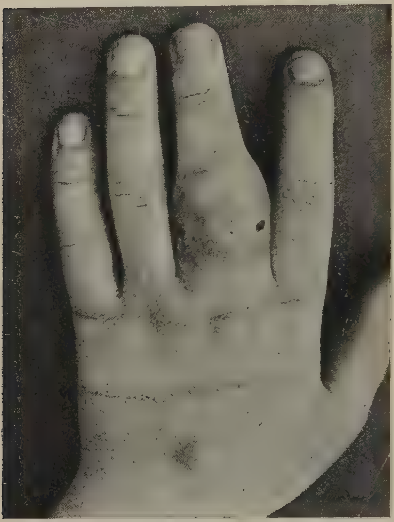

FIG. 147.—Tuberculous disease of proximal phalanx of middle finger.

Prognosis.—In early cases, the disease may subside under treatment, but in cases of extensive involvement the result leaves much to be desired. The end results are a shortened finger and ankylosed joint.

Treatment.—In the early stage, the finger and hand should be immobilized, but if the disease has progressed, and perforation threatens, the diseased tissue should be curretted with a sharp spoon. In order to prevent spontaneous fracture of the affected bone a fenestrated plaster cast is applied. To avoid the tendons and nerves, the incision on the skin is made laterally. If the joint is involved, amputation is indicated.

CHAPTER CXI

BONE AND JOINT SYPHILIS

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Bone and joint syphilis has been known for a long while. Syphilitic arthritis was described as early as 1488 by Petrus Martyr. The condition was also referred to by Alphonsus Ferrus in 1537, and by Morgagni and Fabre in the seventeenth century. Although Hunter did not think that syphilitic arthritis occurred he was opposed by others of his time. Richet, in 1853, was the first to give a clear account of the disease. Since that time the condition has been recognized and has been described by a number of authors, and there is voluminous literature on the subject. In spite of this, however, syphilis does not seem to have been regarded as of much importance or of great prevalence until within the last 8 or 10 years. Since then, however, its rôle in the production of many of the bone and joint lesions has been receiving a great deal of attention. Even at present, the importance of syphilis as an etiologic factor in the cause of bone and joint lesions is not fully appreciated by many.

One cannot read the literature, or have any wide experience with bone and joint lesions, without being impressed by the prevalence of syphilis in these condition, and by the variety and the many ways in which it manifests itself. Bone and joint syphilis may manifest itself in almost any form, may simulate almost every disease and frequently various strains and injuries, so that a mistake in diagnosis may very easily be made, especially if one is not looking for syphilis.

Although the present chapter is intended to deal primarily with congenital syphilis of the bones and joints, it seems advisable to touch also upon the manifestations of acquired syphilis of the bones and joints.

Occurrence.—It is difficult to give exact figures on the occurrence of bone and joint syphilis, as many cases have undoubtedly been erroneously diagnosed. If one depends upon the Wassermann for a diagnosis this error is increased, as this may often be negative, especially in congenital syphilis. The general consensus of opinion at present, is, that bone and joint syphilis is much more frequent than was formerly supposed and that it is probably one of the most common forms of bone and joint lesion.

Higgins states that from 39 to 63 per cent. of congenital syphilitics have arthritis, and that 7 per cent. of the joint lesions of children are due to syphilis.

The author found that out of 619 patients treated in the Orthopedic Clinic of the Washington University Medical School in one year, 10 per cent. had syphilitic arthritis and that 3.8 per cent. of all these cases gave a positive Wassermann. This is undoubtedly a low percentage, as a number of cases were probably overlooked before the investigation began. The percentage of positive Wassermann reactions is also too low because it was not made in a number of cases. These statistics are for adults. At the Clinic of the Children's Hospital, 10 per cent. of all the orthopedic cases seen in one year were diagnosed as syphilitic arthritis. If those cases which were not joint infections, were eliminated, the percentage reached 25. In both these sets of statistics bone syphilis was not included, which of course would make the percentage higher. In both the clinics the percentage of syphilitic arthritis was the same. This does not agree with the statements of other investigators who claim that congenital syphilis is more common.

Bone and joint syphilis may appear at any age. In the author's series of adult cases it was most common between the ages of 31 and 40. In congenital syphilis it is most common in early infancy and again at the end of the first decade and the beginning of the second. It may, however, appear at any age and cases of congenital syphilis of the bones and joints have been reported in adults.

The sexes seem to be affected about equally. Color, probably, does not have much influence. In the author's series 42 were white and 22 were colored. This is about the proportion of the patients seen in the clinic.

Joints Involved.—All the joints and bones may be involved. The knee in both acquired and congenital syphilis seems to be the joint most frequently attacked. Multiple joint involvement is very frequently seen. This tendency to multiple involvement might almost be said to be the rule, if there can be any rule in describing bone and joint syphilis. Syphilis of the spine seems to be uncommon. Aimes suggests that this belief, however, is probably due to blunders in diagnosis. The tibia, of the long bones, is by far the most frequently involved.

Classification.—A classification of bone and joint syphilis is difficult, as there are many manifestations of the disease that do not fit into any classification. The following is suggested, however, as useful in describing the various types seen.

Bone and joint syphilis may be classified as follows:

1. Acquired 2. Congenital

1. Acquired
    - (*A*) Early secondary (Multiple arthralgia).
    - (*B*) Secondary (Synovitis with hydrops).
    - (*C*) Tertiary (Gummatous).

2. Congenital
   (*A*) Early.
   (*B*) Late.

Acquired Syphilis.—(*A*) Arthralgia is frequently noted in the early stages of acquired syphilis. It occurs early, sometimes before the rash, and it is doubtful whether it is a joint or a peri-articular infection. The pain may be intense, in and around the joint, and is often worse at night. It is not increased by motion. The range of motion is not impaired, or is only slightly limited. There may be a slight stiffness in the morning. Several joints may be affected at the same time. These symptoms are similar to those of eruptive fevers. There may be a swelling of the synovia or neuralgic pains, which may be affected by the weather.

BONE INVOLVEMENT.—In this early stage there is very little or no bone involvement, at any rate it has not been noted by most observers. Wile and Senear, however, state that there may be bone involvement in very early syphilis and the periostitis is probably the most common type objectively.

(*B*) During the secondary stage a common form is an acute or chronic synovitis. In many cases it comes on without any warning, usually accompanied by some pain and tenderness, which is increased by motion, and may be unaccompanied by any other signs or symptoms. There is no constitutional disturbance. but in some cases it may be preceded by some malaise.

There is another type that may closely simulate rheumatic fever. It has been described as syphilitic rheumatism. It may be accompanied by a high fever which may be prolonged, but there is no cardiac involvement, nor are there acid sweats. Several joints may be affected at the same time. It may resemble gonorrheal rheumatism. The knees, shoulder, elbow and sternoclavicular joints are probably most often involved.

Hydrarthrosis may appear during the secondary stage. Hydrops may be the only clinical symptom. It may occur in the beginning of the secondary stage or a year or more after infection. Its onset is slow and insidious, usually without pain. The knee is most often affected. It may succeed the more acute synovial effusion.

Bone Changes.—Periostitis is the bone change most frequently seen in this stage.

(*C*) A gummatous osteitis situated near a joint may result in a secondary arthritis, which may closely simulate tuberculosis of that joint. The pain, however, is generally much less than in tuberculosis.

Subacute or chronic synovitis is also met with in tertiary syphilis. The knee is most often affected. There is little pain or interference with motion. There is usually a little thickening of the synovial membrane, and a little creaking. The amount of fluid present may vary. Occasionally there is a slight rise in temperature. The synovitis may appear a number of years after the initial lesions without any other active symptoms and may simulate rheumatoid arthritis.

There is also a form of gummatous synovitis resembling a subacute synovitis, in which subsequently a gummatous mass may be felt. There may be no fluid, but a very irregular lumpy thickening of the synovia. The amount of pain varies, and is frequently greater at night. Motion may cause little pain and there may be very little limitation of motion. There is no starting pain. Remissions and almost complete cure with relapse are very characteristic. It may be mistaken for tuberculosis.

Waterhouse describes a type, which he says is very characteristic, in which the surface of the inflamed joint is covered by circular and serpiginous ulcers. It is seen especially in women rather past middle age.

Wiggs describes a perisynovial hyperplasia, which may terminate in an osteitis of the degenerative type and occasion considerable destruction of tissue and loss of function.

Morestin describes a form of arthritis in which the bone changes are most marked. In this there are marked hyper-ostoses, whose development may be rapid. Pain, if present, is more marked at night. This type may produce ankylosis or marked relaxation.

Intermittent hydrops is also seen in this stage.

Types similar to arthritis, osteo-arthritis, and arthritis deformans are seen at this period. They may be due to syphilitic toxins, or possibly they may be due to the direct invasion of the spirocheta, especially in some of the monarticular types. There may also be a spondylitis of syphilitic origin. Painful feet, simulating flat foot. have been reported and the author has also noted this condition.

Bone Involvement.—Periostitis is one of the marked types of bone involvement in this period. It is practically always multiple and may vary from a slight periosteal elevation to a degree which closely resembles osteomyelitis.

Osteitis is another type of bone involvement. The bone becomes dense and sclerosed.

Gummata of the bone occur at this time. They may be either single or multiple. When they occur near, or in the epiphysis, they may extend into the joint, causing the joint lesions that have already been mentioned.

Congenital Syphilis.—(*A*) Osteochondritis is an early form of congenital syphilis. The onset may be sudden with some constitutional symptoms. Swellings appear at the epiphyses as small hard tumors or general enlargements. They are usually sensitive. Several epiphyses may be involved, usually those at the distal extremities of the bones of the lower legs. At times, the pain and discomfort may cause an apparent paralysis, the so-called pseudoparalysis of Parrot. Again, the joint becomes involved and a secondary synovitis develops, which may become purulent and suppuration result. At other times there may be only a slight synovitis, or no clinical signs.

A simple symmetrical synovitis of the knees is often seen in early syphilis. This condition is also seen in older children between 8 and 14. There is practically no pain and little interference with function. The amount of fluid may be considerable, but there is no other change in the joint.

(*B*) In later syphilis there may be an osteo-arthropathy of a single joint, commonest in the larger joints. It begins insidiously with severe and persistent nocturnal bone pain. Limitation of motion is mechanical. The condition simulates tumor albus.

There may also be a primary gummatous affection of the synovial membrane similar to the condition seen in acquired syphilis.

A deforming type of osteo-arthropathy has been described in which an epiphysis may become irregularly enlarged and greatly changed in shape, with resulting marked limitation of motion. There may be even partial ankylosis, due to alteration of the bone ends. The condition is sometimes mistaken for dislocation. The shoulder, elbow and knee are most often affected.

Bone Involvement.—In the cranium characteristic changes take place. There may be delayed or premature closure of the fontanelles. There may be a thickening of the parietal and frontal bones in the region of the anterior fontanelle, giving a square appearance to the head, "the hot cross-bun head."

The bone manifestations in congenital syphilis are virtually the same as those seen in the tertiary type of acquired syphilis. In addition to these there is also an osteo-chondritis and an epiphysitis. The latter types usually result in secondary joint involvement.

Dactylitis may be mentioned as a special type of bone involvement seen in congenital syphilis. It is found most frequently in the metacarpal or metatarsal bones and begins either in the subcutaneous tissues or as an osteomyelitis in the interior of the bones.

It will be seen that bone and joint syphilis, especially joint syphilis may manifest itself in a number of ways. These may, however, be briefly summarized as follows:

The lesions of acquired and congenital syphilis are to a great extent similar in type. In the early stages they manifest themselves as a simple synovitis which is probably toxic in origin. Later there may develop a simple arthritis secondary to a bone involvement, usually gummatous. The gumma may break into the joint cavity, causing destruction of the cartilage and the joint surface and involving the synovia and the periarticular structures, giving a picture that closely resembles tuberculosis.

The bone lesions are periostitis, osteitis and gumma.

The above are fairly definite types, but joint syphilis does not follow the obvious. It may simulate almost every other joint condition, so that one must constantly have syphilis in mind when treating a disease of the joint. This is especially important in cases of obscure origin, or when the course of the disease is not entirely typical of the conditon diagnosed. The importance of this is strikingly illustrated by Roberts, who states that in 226 cases which he considered congenital syphilis the following diagnoses had been made: 81 cases of joint tuberculosis; 14 cases of osteomyelitis; five cases of torticollis; 12 cases of myeloma; five cases of tuberculous adenitis; seven cases of myositis with sacro-iliac strain; 24 cases of arthritis; eight cases of disease of the carpus or tarsus; 16 cases of chronic headache; 10 cases of osteochrondritis of the hip (Legg-Calve disease); one case of lateral curvature of the spine; one case of lymphangitis.

Roberts also strongly emphasizes the fact that syphilis of the spine may closely resemble tuberculosis of the spine, and that an erroneous diagnosis may easily be made.

A case reported by Gaenslen and Thalheimer in which congenital epiphysitis simulated anterior poliomyelitis and was not recognized until other symptoms of syphilis developed a year later, also emphasizes this point.

Pathology.—The pathological changes that take place in bone and joint syphilis are in the great majority of cases primarily in the bones, the joint involvement is usually a secondary involvement or an extension into the joint from the primary focus.

The most common lesion in early infancy is osteochondritis. Sensitive swellings appear at the epiphyseal junction, either as small hard tumors, or as general enlargements, resembling those of rachitis. As a rule several epiphyses are involved. There is a multiplication and an irregularity of the cartilage cells of the ossifying layer and a premature calcification. Necrosis may result, as shown by a zone of hard dry yellow substance in the ossifying layer of the cartilage, about which newly formed bone is softened and in part replaced by granulation tissue. If the disease is progressive ulceration and suppuration may follow. The cartilage may be destroyed and the epiphysis may be separated, causing deformity and cessation of growth. The neighboring joint is usually involved.

In the later stages of congenital bone syphilis there is a syphilitic periostitis, which may involve any of the bones. The tibia is most frequently involved. The periostitis is usually diffuse, involving the entire shaft of the bone. This results in an overgrowth of the cortex on the convex side. In the tibia this usually affects the crest, as a result of the trauma to which this part of the bone is constantly exposed. This results in a hypertrophy of the shaft along the crest, giving the characteristic "saber-shaped" leg. The bowing is always anterior, and is a long gradual curve.

Nodes may develop in the long bones. These are ill-defined swellings adherent to the bone, and unless inflammation takes place the skin moves easily over it. If the node becomes infected it may suppurate, a part of its contents is discharged and an ulcer is formed with a typical wash-leather base. Beneath the ulcer the superficial layers of the bone are often necrosed.

Osteitis and Osteomyelitis.—Gummatous inflammation may be either localized or diffuse. If it is localized it may so weaken the bone as to cause spontaneous fracture. More frequently the syphilitic process causes a general sclerosis throughout the bone so that it is thickened and heavier than usual. As a rule, it does not go on to suppuration and rarely to necrosis. The spongy bones are often affected by osteomyelitis.

Dactylitis begins either in the subcutaneous tissue or in the interior of the bone as an osteomyelitis. As a rule, it does not break down unless the tissues become infected with organisms.

Joint Syphilis Acquired.—Comparatively little is known of the pathologic conditions present in the arthralgias and arthritis of secondary syphilis, due to the fact that autopsies have not been secured. The arthralgias are probably due to a toxic irritation, similar to those seen in the infectious fevers. In the later secondary stage the lesions consist mainly of synovitis and tend to heal without impairment of function, or without any permanent tissue change. The lesions are probably the same as in the earlier stage, but vary with the intensity of the symptoms. There is usually some moderate thickening of the synovial membrane. The amount of the fluid may vary from a small quantity to a large amount.

The lesions of tertiary syphilis are much more extensive. The synovitis seen in this stage may be due to toxic irritation, or it may be the result of gummatous involvement of the synovial membrane. At times this gummatous mass may be so extensive as to fill the entire joint, and it may very closely resemble tuberculosis. The process may extend to the bone. If the process progresses too far, extensive damage may result. When the gumma is primary in the bone or cartilage the destruction is much greater.

When the syphilitic lesion is in the bone it is in the form of a gummatous deposit, with syphilitic osteitis and enlargement of the bone. As it progresses it may open into the joint through the cartilage. When the process starts in the cartilage it has been described as chondritis syphilitica. In this condition there is a loss of cartilage accompanied by fibrous scarring. This destruction does not appear at the point of greatest pressure, as is the case in osteo-arthritis, but frequently it is to be seen as a circular pit-like depression. If a fibrous change has occurred fibrillation is not present, but stellate scars, which are characteristic.

Gummatous osteitis, when situated in the neighborhood of the joints, is a cause of secondary arthritis. On section, the bone shows most often, in the periphery, a yellowish-grey focus of disease, in appearance strikingly like the early stage of focal tuberculosis. But from this latter it may be distinguished by the absence of any surrounding hyperemia, or infiltration, which goes with tuberculous disease.

The pathologic changes in syphilis and tuberculosis may be so similar that at times it is very difficult to differentiate between them, even under the microscope. This is illustrated in one of the author's cases.

A colored patient had an infection of the elbow. An abscess developed which had to be opened. At the operation, a piece of bone and some of the necrotic material were removed for examination. The pathologist's report was as follows: "Small pieces of bone and peri-articular tissue. Microscopic examination: Necrotic areas, surrounded by connective tissue, infiltrated with epithelioid cells, and by lymphocytes, are seen throughout. An occasional giant cell is seen. Muscle and bone in some areas is seen, infiltrated by cells. Diagnosis: Tuberculous osteomyelitis." This patient got completely well under anti-syphilitic treatment. She had a four plus Wassermann. Some of the material injected into a guinea pig gave negative results.

Hereditary Syphilis.—In congenital syphilis, as in acquired syphilis, the joint changes are, as a rule, secondary to bone syphilis in their neighborhood. These, in the milder cases, may be a simple serous hydrarthrosis. In the more severe types the involvement may be so great as to invade the joint by direct continuity, or by eruption of pus into the joint cavity.

In the joint condition associated with epiphysitis, the joint seems to be filled with a soft fungus substance.

Hydrops is an important type. The synovia is thickened locally and diffusely.

There are two forms of gummatous joint involvement, one, synovial or capsular, that spreads into the articulation and causes villi which may contain gummata; the other is primarily osteal, extending into the joint, causing extremely destructive changes in the bone and cartilage, and in some cases, resulting in suppuration and ankylosis.

A condition occasionally occurs in which the epiphysis becomes deformed in an irregular manner by osteophytic outgrowths. There results marked deformity in the joint and much limitation of motion, or even partial ankylosis, due to alterations in the bone ends. There may be muscular and ligamentous contractures.

In older children, about 14, a rarefying osteitis, associated with a deposit of caseous material in the cancellous tissue, affects many bones. The process extends to the articular cartilage, causing a grooving and pitting, the condition known as chondro-arthritis. This form is said to be resistant to treatment and practically incurable.

It will be seen from the above that the predominating feature of bone syphilis is a periostitis and osteitis. Hyperplasia of bone is always present and the bone lesions are usually multiple.

In joint syphilis, synovitis without marked change marks the early stages. In the later stage the changes are primarily gummatous, accompanying which there may be marked destruction of the joint structures. This is usually associated with bone change and is frequently secondary to bone involvement.

Trauma seems to play an important rôle in the localization of the lesion, especially in acquired syphilitic arthritis. There is apparently a very definite relation between the occupation of the patient and the joint involved, which would tend to show that a joint subjected to constant use and the trauma of occupation tends to develop syphilitic arthritis. This agrees with the experience of other writers on the subject.

X-ray.—The *x*-ray has played an important rôle in our study of bone and joint syphilis. It has also been of great value in furthering our knowledge of the pathological changes that take place. It is much more useful, however, in the study of the bone changes than in the articular changes.

Periostitis, either diffuse or local, is accompanied by the formation of an osteoid material, that shades off in appearance from a gelatinous substance in new deposits to a heavier or calcareous appearance in the older deposits. This deposit in layers is probably due to successive attacks of periostitis. A new layer is probably laid down in each succeeding attack. With a high-power hand glass the appearance of the topmost layer is that of a feathery edge. Here and there over different parts of a bone there may be isolated patches beneath the periosteum, raising it from the bone cortex. A marked circumferential thickening of the periosteum is a typical finding in bone syphilis. The condition may vary from a slight periostitis to an advanced osteomyelitis. As a rule, the calcium salts are laid down more or less parallel to the long bone and are frequently raised a little from the shaft. This type cannot be differentiated from the inflammatory type. Occasionally the calcium salts may be laid down perpendicular to, and extending out from the shaft about ¼ in. This may, for want of a better name, be spoken of as the "lace-work type" of periostitis, and when this is present it is invariably syphilitic in origin. Unfortunately this type is the exception rather than the rule.

Osteitis may be either diffuse or local. In very early cases there may be no involvement of the cortex shown. A characteristic osteitis is present in the majority of cases at a later date. This is marked by a thickening of the cortex or of both the cortex and the spongeoid bone. The process is a building around of one layer upon the other. In osteitis the differences in structure perceptible in normal bone become less distinct and the bone seems denser (sclerosed). The growth of cortical bone may be either external, producing the so-called bowing of the shaft, or it may be internal, encroaching upon, or obliterating the medullary canal. As the process progresses the bone and periosteum may become fused into one mass. Gumma formation generally accompanies osteitis when the process is severe. They are usually single, but may be multiple. They may be very small or involve enough of the bone thickness to cause pathological fracture. As the osteitis goes on areas of necrosis may occur, surrounded by areas of greater bone activity. These appear on the *x*-ray as areas of translucency surrounded by areas of osteosclerosis.

Typical acute gummata are easily recognized. They have generally a translucent center with destruction of bone; immediately surrounding this central mass there is a dense sclerosed wall, and over this generally an area of periostitis. In chronic gummata there is marked sclerosis with localized swelling, destruction of bone usually being present.

The author wishes to emphasize again the fact that bone and joint syphilis may manifest themselves in a great variety of ways, and that they may simulate almost every variety of disease of these parts. Unless this fact is clearly borne in mind many mistakes will be made.

Symptoms.—The symptoms of bone and joint syphilis are almost as varied as the types, and very much more contradictory. As would be expected they vary with the lesions which they simulate.

Onset.—The onset is usually insidious. It may, however, be sudden, accompanied by marked constitutional symptoms. In the early stages of acquired syphilis several joints may be affected simultaneously or in rapid succession before the infection finally settles in one or more joints. Trauma and occupation seem to have a determining influence on the location of the lesion and the joint involved.

Pain.—Pain is a variable symptom. Its manifestations are very contradictory. It may or it may not be present. It may be severe or moderate. In bone syphilis pain is usually severe and is, as a rule, worse at night. In joint syphilis pain is not as severe, and it seems to be less marked than in other lesions in which there is a similar amount of joint involvement.

Some writers believe, however, that pain is a predominating feature. Wallace states that osteocopic pain was present in a severe form on 56 per cent. of his cases of acquired syphilis. In 19 per cent. of the congenital cases it was present, but was not very severe. In the remaining congenital cases there was no osteocopic pain.

Exacerbations of pain at night are supposed to be typical of syphilis, and to be an almost diagnostic sign. This is probably true in some of the bone lesions, but according to the author's experience is not necessarily a feature of joint syphilis. In 57 per cent. of his series of cases pain was most marked in the day time. In the late stages night exacerbations seem less frequent than in the early stages. An increase of pain at night, however, should be considered a very significant sign.

In the early stages before destruction has taken place, the pain is not greatly affected by motion. This, however, is not an invariable sign. Later, with destruction of cartilage there may be marked pain on motion.

Indefinite joint pains are characteristic and suggestive.

Tenderness.—In periostitis the tenderness may be very acute. In arthritis there is usually tenderness present. This may or may not be marked.

Muscle Spasm.—Muscle spasm is usually slight or absent, though here again there are numerous exceptions, especially in the more severe types, accompanied by joint destruction.

Swelling.—In periostitis of the milder types swelling may be slight or absent. In the more severe types of periostitis and in gummatous involvement of the bone there is usually swelling of the adjacent soft tissue and on palpation, osseous thickening may also be made out. In the simple synovitis there is, of course, swelling of the joints, due to the increase of fluid in the joint. In arthritis and gummatous synovitis there is swelling of the joint due to increase of fluid and to peri-articular thickening, and in those cases where the condition is secondary to a bone lesion there may also be made out osseous thickening. In the arthralgias there are no joint signs. Fluctuation is as a rule, not present.

Limitation of Motion.—Motion is not markedly limited in the simple types of arthritis. In those cases where there has been marked joint destruction, motion may be markedly limited, or even absent.

Redness and local increase in temperature are not common, but may occur.

Fever is comparatively rare, but in some cases it may be present, especially in the secondary stages and in the acute types of congenital syphilis. It may be very high at times and may simulate acute articular rheumatism.

Atrophy is usually slight or absent.

Suppuration is not common, but may occur.

There may also be numerous vague and indefinite symptoms, especially pain, which do not seem to belong to any definite joint lesion.

It will be seen from the description of the symptoms of bone and joint syphilis that they are all rather indefinite. All the symptoms enumerated: Pain, tenderness, muscle spasm, swelling, limitation of motion, redness and local increase in temperature, fever, atrophy, suppuration, and numerous vague and indefinite symptoms may or may not be present, and all vary in intensity from very slight or absent to very severe. The only possible exception to this is swelling, which is practically always present to a greater or less extent, especially in the joint lesion.

The author believes that this very atypicalness of the symptoms is an important characteristic of bone and joint syphilis and that it is almost a diagnostic sign. He believes that in all joint lesions in which the symptoms are atypical, syphilis should be suspected.

Diagnosis.—It is most important that a correct diagnosis should be made in syphilis of the bones and joints, because the treatment in these conditions is radically different from the treatment in other joint lesions. The diagnosis is not always simple and is based on the following points: History, physical signs and symptoms, *x*-ray, Wassermann, and therapeutic test.

History.—In all cases a very careful history should be made and especial attention should be given to the family and the past history. In adults one must consider the possibility of either acquired syphilis or possibly a late manifestation of hereditary syphilis. All points that might suggest the possibility of a syphilitic infection, either in the patient or in the parents, should be elicited both by direct and indirect questioning. One should not fail to inquire about the previous health of the parents; the number of miscarriages, if any should be noted, and the possible cause; the number of still births is also significant. The number of children dead, if any, and the cause of death; the general health of the living children. A history of chronic sore throat or skin lesion in the parents or the patient should arouse suspicion, as would also the history of nervous disorders in the parents or patient. A direct history of venereal infection is of great value, but a denial of infection does not exclude the possibility of its existence. In many cases a history of syphilis is not obtained, even when the patient is perfectly honest, and does not desire to conceal the truth. Frequently, the site of infection is so slight or its location such that it has escaped the patient's attention. The secondaries, if there were any, have also been overlooked by the patient. This fact may be illustrated by a brief summary of a case, which will also illustrate the subtle ways in which joint syphilis may manifest itself.

A man 35, single, was seen because he complained of painful feet. On general examination he seemed normal except for flat feet. There was no history of venereal infection. He was given treatment for his flat feet and was told to return in a week. When he returned his feet were still painful and he also complained of pains in his shoulders, arms and back. Because of the indefinite pains and the failure of improvement in the foot condition a suspicion of syphilis was aroused. The patient was again asked whether he had ever had syphilis or other venereal infection. He repeated his previous denial, evidently quite sincerely and honestly. He was advised to have a Wassermann test. This was reported to be four plus. The patient was astounded, especially as he was to be married in three or four weeks. On his again being questioned, he remembered that about 10 or 15 years before he had had a slight abrasion that he had entirely forgotten. Under the circumstances, he was sent to a dermatologist for treatment. Another Wassermann was made by a different serologist and again the Wassermann report was four plus. Under the treatment for the syphilis the foot pains and other symptoms steadily improved.

A history of soft chancre, gonorrhea or even a simple abrasion or cut is suspicious and should lead one to suspect the possibility of syphilis, and to make a thorough investigation.

Physical Signs and Symptoms.—The physical signs and symptoms in bone and joint syphilis are rather indefinite and contradictory, as has been pointed out. For that reason they are not of great value unless supplemented by the other diagnostic points. Pain, which is worse at night or not commensurate with the amount of bone or joint involvement is suggestive, as is also pain not affected by motion; absence of marked limitation of motion, muscle spasm or atrophy are suggestive of syphilis, also multiple joint lesions. When these symptoms, or others not typical of any other symptom complex, are met, they should immediately arouse the suspicion of syphilis.

In congenital syphilis a diagnosis is more readily made on the physical signs than in acquired syphilis. Here, in addition to the actual bone and joint involvement, there are usually other signs of congenital syphilis, which must be carefully sought after. Among these may be mentioned: Cicatrices about the mouth and anus, snuffles, adenoids, which may at times suggest syphilis, high arched palate, Hutchinson's teeth, nervous symptoms and others.

Painless bilateral effusion of the knee joint is very suggestive of hereditary syphilis.

X-ray.—The *x*-ray is of great value in the diagnosis of bone and joint syphilis, especially of the osseous type. The bone manifestations of syphilis are practically always multiple. Periostitis, which is most common, may be found involving a number of bones in a greater or lesser degree. In practically all the joint lesions there is also bone involvement. In a joint lesion in which there may be doubt as to the diagnosis and where syphilis is suspected several bones should be *x*-rayed. If the condition is syphilitic, periostitis or other bone involvement will probably be seen.

In joint syphilis the *x*-ray does not show any very characteristic joint changes. These must be sought in the adjacent bone.

The most important and characteristic changes shown by the *x*-ray are:

(*a*) Periostitis.
(*b*) Osteitis.
(*c*) Gummata.
(*d*) Osteochondritis.

(*a*) Periostitis may show as a small localized area on the bone, or it may be diffuse, involving the entire shaft. In appearance there may be a simple thickening and roughness along the edge of the bone in the milder types. In the more severe and advanced cases there is marked periosteal thickening and raggedness. It may have a moth-eaten appearance. At times there may be layers of calcareous deposit, probably due to successive attacks of periostitis. In spots the periosteum may be raised from the cortex. This is usually due to subperiosteal gumma. A marked circumferential thickening of the periosteum is a typical finding in bone syphilis. Baetger and Waters speak of the "lace-work type" of periostitis, which, when it is present, is invariably luetic in origin.

(*b*) Osteitis first shows as a thickening of the cortex, usually on one side. Later the cortex on both sides of the bone, as seen on the plate, may be thickened with a corresponding decrease in the size of the medullary cavity, or even complete obliteration. At the same time the bone appears denser and the normal markings are less distinct, and may finally disappear. In some cases this cortical deposit may be external, appearing along the exposed borders of the long bones, such as the crest of the tibia. The deposit may be sufficient to cause an apparent bowing of the bone. In these cases the edge of the bone is rough and irregular. As the osteitis goes on, areas of necrosis may occur surrounded by areas of greater bone activity. These appear on the *x*-ray plate as areas of translucency surrounded by areas of osteosclerosis. Gumma formation generally accompanies osteitis when the process is severe.

(*c*) Typical acute gummata are easily recognized. They have generally a translucent center with destruction of bone; immediately surrounding this central mass there is a dense sclerosed wall and over this, generally, an area of periostitis. In chronic gummata there is marked sclerosis with localized swelling, destruction of bone usually being present. There may also be a gummatous osteomyelitis, which in the *x*-ray closely resembles the ordinary pyogenic osteomyelitis.

(*d*) In osteochondritis the *x*-ray shows a thickening of the bone with a separation of the shaft and the epiphysis.

In bone syphilis there is always formation of new bone.

In syphilitic arthritis there is no characteristic *x*-ray picture. This may be similar to tuberculosis or any other joint infection. There may be little joint involvement, or there may be marked destruction of cartilage and bone. A clue to the condition is usually found in the discovery of bone lesions, either adjacent to the joint or in some other bone.

Wassermann.—A positive Wassermann is strong presumptive evidence that the bone or joint lesion is probably syphilis. It must be remembered, however, that tuberculosis and syphilis are often associated, or that, as one may have syphilis, and a joint lesion that may be the result of some other type of infection, a positive Wassermann does not always mean that the joint is necessarily syphilitic.

A negative Wassermann does not exclude the possibility of syphilis. This seems to be agreed upon by most of the writers on the subject. Coues expresses the opinion on this subject very aptly. He states: "The Wassermann reaction is a mixed blessing in the diagnosis of syphilis." Despite much clinical evidence, and much writing, many physicians, specialists, and general practitioners alike, still continue to believe that in an obscure case, a negative blood Wassermann reaction rules out syphilis.

Jeans, however, states that the Wassermann reaction is always strongly positive in congential syphilis in younger children and unless this is the case a diagnosis of congenital syphilis should not be made.

In congenital syphilis the Wassermann test is much more frequently negative, especially in older children. Consequently a negative Wassermann must not influence one in excluding syphilis, unless one is sure after a careful study of the other points of diagnosis that syphilis is not present. If the Wassermann test is negative and there is any doubt, it is advisable to have a Wassermann test on one or both parents. If there is a negative Wassermann in a suspected case the patient should be given anti-luetic treatment and the Wassermann repeated.

The method employed in making the Wassermann test is often of importance. Serologists of experience agree that the blood of syphilitic children often fails to give a positive reaction with alcoholic antigens, and that even with the use of cholestrin negative results are not infrequent.

The luetin test may be an aid in diagnosis.

Therapeutic Test.—In cases in which a diagnosis is difficult to make, and where the possibility of syphilis is suspected, the therapeutic test should be used. In many cases of bone and joint syphilis the results obtained by the use of anti-syphilitic treatment are almost like magic. In these doubtful cases the results of treatment can be followed not only by the improvement in the clinical signs, but also by the improvement in the pathological condition as demonstrated by the *x*-ray. Under these circumstances a diagnosis of probable syphilitic infection is permissible.

The points, then, upon which a diagnosis is made may be briefly stated as follows:

(a) **History.**—A very careful history should be made, and all previous venereal infection investigated. If there is no history of venereal disease suspicious signs should be sought, either in the patient or in the family history.

(b) **Physical Findings.**—Swelling of bones or joints usually multiple. Pain, usually less severe than would be expected with the amount of joint or bone involvement, which may or may not be worse at night. If worse at night it is quite suggestive of syphilis. Limitation of motion is usually absent or slight. Muscle spasm is usually absent. The symptoms may be contradictory, and if a-typical should arouse a strong suspicion of syphilis.

(c) **X-ray.**—Periostitis is almost always present and usually involves a number of bones. Osteitis is frequently present. It shows a thickening of the cortex and a blurring of the normal bone shadows. It may cause a bowing of the bone on one side. Gummata show up as a destructive process surrounded by a wall of sclerosis. Osteochondritis begins at the diaphyso-epiphyseal line, usually on the diaphyseal side and may extend to the joint, causing joint destruction. In pure joint syphilis the *x*-ray may not show any typical signs, but an examination of other bones may clarify the picture by showing typical periostitis.

(d) **Wassermann Test.**—A positive Wassermann test points strongly to syphilis though there may be a mixed infection, usually tuberculosis, or there may be a joint lesion in addition to syphilis. A negative Wassermann does not exclude syphilis, especially in the congenital type. When negative a careful search should be made for other signs of syphilis, either in the patient or in the parents. If there is any doubt a Wassermann should be made on one or both parents.

(d) Therapeutic Test.—When in doubt the therapeutic test should be employed, followed by repeated Wassermann tests.

Differential Diagnosis.—As has been stated, syphilis may simulate almost any type of bone or joint lesion. The most important ones, however, from which it must be differentiated are:

(*a*) Tuberculosis of the bones.
(*b*) Tuberculosis of joints.
(*c*) Tuberculous dactylitis.
(*d*) Rickets.
(*e*) Scurvy.
(*f*) Synovitis.
(*g*) Osteomyelitis, infectious.
(*h*) Typhoid osteomyelitis.
(*i*) Arthritis, acute.
(*j*) Arthritis chronic.
(*k*) Arthritis, deformans.
(*l*) Still's disease.
(*m*) Gonorrheal rheumatism.
(*n*) Erbs paralysis.
(*o*) Anterior poliomyelitis, acute.
(*p*) Sarcoma.
(*q*) Carcinoma.
(*r*) Bone abscesses.

(a) Tuberculosis of the Shaft of the Bones.—The onset may be more or less similar. Pain in Syphilis is worse at night. There is less atrophy in syphilis. There may be a history of syphilis or other signs of syphilis.

X-ray.—In syphilis the lesion begins in the epiphyseal end of the diaphysis or in the shaft, while in tuberculosis it begins in or near the epiphysis. In tuberculosis the bone is destroyed just as in ordinary osteomyelitis, and there may be a marked periosteal bone production, but seldom new bone in the shaft. The lesion, in tuberculosis, is frequently multiple, as in syphilis. It seems almost impossible to differentiate tuberculosis from syphilis.

In syphilis the Wassermann test may be positive and in tuberculosis there will probably be a strong von Pirquet reaction. There will also, probably, be other signs of syphilis. If the diagnosis cannot be established the therapeutic test should be tried.

(b) Tuberculosis of the Joints.—*History.*—There may or may not be a history of tuberculosis or syphilis. Careful questioning, however, will probably elicit some history of syphilitic infection.

The onset is usually similar, more or less insidious. Pain in tuberculosis is severe and is increased by motion. Night cries are usually present. In syphilis the pain is much less marked, or absent, and as a rule is not affected or very slightly increased by motion. Night cries are usually absent. In tuberculosis there is atrophy and muscle spasm; in syphilis there is little atrophy or muscle spasm. In tuberculosis there are no night exacerbations of pain; in syphilis there may or may not be. In tuberculosis the temperature variations are characteristic; in syphilis there may be a general rise in temperature or it may be normal. In tuberculosis, abscess formation is more frequent than in syphilis. In tuberculosis the joint involvement is practically always monarticular; in syphilis it is more likely to be poliarticular.

X-ray.—In tuberculosis the process begins in the epiphysis, in syphilis it begins in the epiphyseal end of the diaphysis. In syphilis there is marked periosteal thickening, in tuberculosis there is none. In syphilis there is bone proliferation, in tuberculosis there is bone destruction. In syphilis there is hypertrophy, in tuberculosis there is atrophy. In syphilis the swelling is due to thickening of the bone, in tuberculosis it is due to thickening of the soft parts. In syphilis the *x*-ray picture is clear, in tuberculosis it is hazy. In some of the types of syphilitic arthritis, especially when the gummatous process begins in the synovia, the differentiation between tuberculosis and syphilis may be impossible. In these cases it is advisable to take *x*-rays of the other bones to see whether they show periostitis or other signs of syphilitic bone involvement. It must be remembered that tuberculosis and syphilis are frequently associated, and in these cases the *x*-ray picture is especially confusing.

A positive Wassermann test and the therapeutic test, especially when combined with a strong tuberculin reaction, will help clear up these combined cases.

(c) **Tuberculous Dactylitis.**—This condition is more common than syphilitic dactylitis. It usually involves the phalanges and most frequently the first phalanx of the index finger. It occurs most often in early life during the second and third years. In syphilis the condition is generally multiple and is said to affect the metacarpals and the metatarsals almost entirely. Suppuration is less common in syphilis than in tuberculosis.

X-ray.—Tuberculosis originates in the epiphysis, and syphilis in the epiphyseal end of the diaphysis. In tuberculosis, periosteal thickening is absent or nearly so, in syphilis it is marked. Tuberculosis has a greater tendency to bone destruction, syphilis to bone proliferation. Inflammation of the soft parts causes swelling in tuberculosis, in syphilis it is largely due to thickening of the bone.

(d) **Rickets.**—Rachitic bone lesions usually do not appear before the period of the first dentition, while in syphilis they appear in the fetus or the newborn. In Rickets there is a multiple enlargement of the epiphysis with rachitic rosary, in syphilis the ribs are rarely involved. The history and the Wassermann test are of importance. Rickets and congenital syphilis are frequently associated.

X-ray.—In rickets the cartilaginous surfaces are intact, but there is marked disturbance of the epiphyseal line. The epiphyseal line becomes softened and has a tendency to spread out, so that the bone at that point is actually wider than normal. There is slight condensation of the calcium salts at the epiphyseal line and the end of the bone has an inverted saucer-shaped appearance. The epiphysis proper does not seem to be disturbed. The changes are confined entirely to the epiphyseal line. The saucer-shaped expansion is most marked when the stress and strain is directed perpendicularly to the epiphyseal line. In rickets there is bone atrophy, in syphilis there is none. In syphilis the bowing of the tibia, "saber leg deformity," is due to the deposit of bone along the crest of the tibia, but in rickets the curve is due to softening of the bone and to actual bending of the bone. In syphilis the curve is an apparent one, in rickets it is an actual one. Syphilitic bone enlargements have a tendency to involve the shaft, by periosteal implication, in rickets the enlargement is usually limited to the epiphysis.

(e) **Scurvy.**—Scurvy usually occurs in late infancy. The feeding history is suggestive. Tenderness and swelling are marked and the gum changes are characteristic. The joint involvement is usually multiple. Peri-articular swelling is generally present.

X-ray.—The cartilaginous surfaces of the joint are intact, but, as in rickets and syphilis, there are marked disturbances in the zone of the epiphyseal line. All the changes, however, take place upon the diaphyseal side of the epiphyseal line. The epiphysis and the epiphyseal line are intact, no changes at all take place in these two structures. There is no saucer-shaped expansion as in rickets, nor localized areas of softening and destruction as in syphilis. Just behind the epiphyseal line is what looks like a second epiphyseal line, in reality a band of localized destruction about 2 mm. in width, extending through the entire bone and parallel to the epiphyseal line. The edges of this band are denser than normal and give the appearance of eburnated bone (Trummer's Band). Atrophy may be present, but, as a rule, the bone texture appears normal. Periostitis is practically always present and it is quite common to have hemorrhages beneath the periosteum.

The main differential points in the *x*-ray diagnosis of rickets, syphilis and scurvy depend upon the changes in and about the epiphyseal line and the character of the periosteal changes. In rickets the changes are confined to the epiphyseal line, in syphilis the epiphyseal line and the bone behind it are involved, while in scurvy the epiphyseal line is intact, and all the changes, comprised in a zone of destruction, take place just behind the epiphyseal line.

In rickets there is seldom periostitis, in syphilis there is marked periostitis, in scurvy the periostitis is frequently accompanied by subperiosteal hemorrhages. In rickets there is atrophy, while in syphilis and scurvy there is little or no atrophy.

(f) **Synovitis.**—A diagnosis between a simple synovitis and a syphilitic synovitis is frequently very difficult. It is based on the history, the *x*-ray, in which signs of bone changes may be found suggesting syphilis, on the Wassermann test and if necessary, on the therapeutic test.

(g) **Infectious Osteomyelitis.**—In osteomyelitis the onset is usually more acute, though there may be an acute onset in syphilis. The temperature in osteomyelitis and the constitutional reaction is greater than in syphilis. In osteomyelitis there are definite signs of inflammation, while in syphilis these are generally lacking. The leukocyte count in osteomyelitis is high, in syphilis there is no change. In syphilis the condition is usually multiple. In syphilis there are usually other signs of syplilis. A positive Wassermann test would be of value. It is possible to have osteomyelitis and syphilis together. In syphilis the bone changes as shown in the *x*-ray are out of all proportion to the clinical picture.

X-ray.—In syphilis the involvement is usually on one side, and is usually a hypertrophy of bone, while in osteomyelitis the shaft is about equally involved in all portions, and the bone density is irregular, showing some localized increase and some localized decrease. Osteomyelitis leads to sequestration, while in syphilis that is rare. Osteosclerosis and osteoporosis are more uniform in syphilis than in osteomyelitis. The new bone formed in osteomyelitis is thinner and more porous and the borders are thinner and more irregular than in syphilis. In chronic pyogenic osteomyelitis there is extensive destruction or absence of bone cortex, and a sequestrum large or small, while this is absent in syphilis. In some cases it is very difficult or impossible to make a diagnosis from the *x*-ray.

The diagnosis should be made on the history, the onset, the physical signs, the *x*-ray, the Wassermann test and, if necessary, on the therapeutic test.

(h) **Typhoid Osteomyelitis.**—Typhoid osteomyelitis simulates very closely the bone changes in syphilis. A very careful history must be made to elicit either a previous syphilitic or a typhoid infection. The Wassermann test is of value. Here the therapeutic test is of great value.

X-ray.—Typhoid osteomyelitis may easily be confused with syphilitic osteomyelitis and a differentiation by the *x*-ray is almost impossible.

(i) **Arthritis of the Joints, Acute, Infectious.**—In acute infections of the joints the onset is more sudden, as a rule, than in syphilis. Acute arthritis is usually monarticular. The constitutional disturbance is greater. The temperature is much higher than in syphilis. Pain and tenderness are much more marked in arthritis. The white count is high in arthritis. There is an increase in the local temperature of the joint and there are other signs of inflammation in acute arthritis. If the process has progressed to suppuration aspiration of the joint will reveal pus, which is usually not found in syphilis. The Wassermann test may also be of value.

X-ray.—In acute arthritis there will probably not be any bone change unless the process has extended to the bone. In syphilis there will be signs of bone change.

(j) **Chronic Arthritis.**—The history is of importance in showing a syphilitic infection, also the presence of other signs of syphilis. The onset and physical signs may be very similar, they both come on gradually and there may not be marked constitutional disturbances.

The temperature may be practically normal in both cases, or only slightly elevated in the chronic arthritis. A positive Wassermann test is of importance.

X-ray.—The *x*-ray in both cases may be very similar, but in syphilis there are usually signs of other syphilitic bone changes.

The therapeutic test is frequently the only deciding point in the differentiation.

(k) **Arthritis Deformans.**—The differentiation may be difficult in these cases, especially as the typical joint changes may be caused by syphilis. The history, the Wassermann test and the therapeutic test will help clear up the condition.

(l) **Still's Disease.**—Still's disease, while presenting some similarity to a syphilitic process in the form of multiple joint involvement, is associated with enlargement of the lymphatic glands, leukocytosis, secondary anemia and constitutional disturbances. There is also marked muscular atrophy. The history, signs of congenital syphilis and the Wassermann test will help differentiate the two conditions. The therapeutic test will also be of great benefit.

(m) **Gonorrheal Arthritis.**—Gonorrheal arthritis may very closely simulate joint syphilis. In the acute type of gonorrheal arthritis the onset is usually more acute and the symptoms much more severe than in syphilis. In the chronic types the two conditions may be very similar. In these cases the history is of great value in showing a previous gonorrheal infection, or the presence of a chronic gonorrhea. It must be remembered, however, that gonorrhea frequently masks a syphilitic infection and that a history of gonorrhea without a syphilitic history does not exclude the possibility of a syphilitic joint. The Wassermann test is of value and where there is any doubt the therapeutic test should be used.

X-ray.—In gonorrhea there are no characteristic changes in the joint. In syphilis there are the characteristic bone lesions.

(n) **Erb's Paralysis.**—The pseudo-paralysis of Parrot may simulate Erb's paralysis. Erb's paralysis is noticed at, or shortly after, birth. The paralysis is characteristic, and there is a history of prolonged difficult labor or of instrumental delivery. In osteochondritis the *x*-ray shows typical syphilitic bone lesions.

(o) **Anterior Poliomyelitis, Acute.**—Poliomyelitis may also be simulated by the pseudo-paralysis of Parrot. The age of onset is suggestive, poliomyelitis is uncommon in children under six months of age, while osteochondritis occurs in early infancy. Paralysis in an infant under six months of age is very suggestive of syphilis. Of value in making a diagnosis are the history and the occurrence of other signs of syphilis, or the exposure to poliomyelitis and its prevalence in the community. An examination of the spinal fluid will show the typical changes if the condition is that of poliomyelitis.

X-ray.—The *x*-ray will show the typical bone changes in syphilis. These will be absent in poliomyelitis.

(p) **Sarcoma.**—In sarcoma the symptoms are more severe, and the pain more constant than in syphilis. The pain is not affected by treatment. The Wassermann test is of importance.

X-ray.—Sarcoma affects the ends of the diaphyses by preference. There is some disturbance in the minute structure of the bone; this consists in an absorption of lime salts, with which may be associated increased density in certain areas. This disturbance is comparatively localized and spreads peripherally, the greatest destruction taking place at the point of origin. Associated with this is a swelling and new growth. In a central sarcoma the walls of the bone seem to be bursting apart, as it were. In peripheral sarcoma the surface of the bone is seen to be rough and uneven and the density shades gradually into the soft tissues. In the peripheral variety the periosteum is often found lifted up, highest over the greatest diameter of the tumor, and then sloping downward gradually until it becomes a part of the bone again.

(q) **Carcinoma.**—Carcinoma of the bones is usually a metastasis and generally occurs in the later years of life. The history and Wassermann test are of value.

X-ray.—In carcinoma there is never any tendency to bone hypertrophy, as one would expect from the pathology. It involves not only the bone substance, but the stroma as well, hence there is an almost complete destruction of the bone substance. The bone substance remaining in the tumor is spongy, porous and eroded.

(r) **Bone Abscesses.**—A simple bone abscess may resemble a gumma of the bone. The differential diagnosis may be made by the Wassermann test and the history, and by the presence or absence of other signs of syphilis. The constitutional signs and symptoms may be very similar.

X-ray.—In the *x*-ray the bone abscesses are seen to be quite large in size and are surrounded by a wall of increased bone density. When they become very large considerable trabeculation may be found in the area involved. This condition is usually not seen in a syphilitic gumma. The gumma is usually multiple.

Prognosis.—Practically all the forms of syphilitic bone and joint disease show marked and rapid improvement under treatment. If the diagnosis is made early and treatment is begun before there has been destruction of bone, there should be little or no deformity or interference with function. In many cases, even where there has been apparently marked destruction, restoration takes place under treatment with marked improvement in function. Unless treatment is continued, however, joint symptoms tend to recur, and where treatment is neglected deformities and more serious complications may result.

Treatment.—A correct diagnosis is absolutely essential for the proper treatment of bone and joint syphilis. This may simulate so many types of bone and joint lesion, that unless the proper diagnosis is made the treatment will vary with the diagnosis, and if the patient is not treated for syphilis the condition will not materially improve, certainly will not be cured, nor is there any condition in which a mistake in diagnosis may do more harm. In modern orthopedic treatment, surgery is playing a constantly greater part. As an example may be cited the use of bone grafts and the various ankylosing operations in the treatment of tuberculosis of the joints. These operations are all intended to destroy the joint and prevent function, a condition most desirable in tuberculosis, but once the desired result has been obtained, function can never again be fully restored. If, as a result of an error in diagnosis, a syphilitic joint is operated upon, a very serious and permanent damage will have been done to a joint which under proper anti-syphilitic treatment could have been restored with complete function. In this case the patient has suffered an irreparable damage as the result of improper diagnosis.

No operation on chronic infections of the bones or joints should be undertaken until after a Wassermann test has been made, and if this is negative, the patient should be given, when possible, a course of anti-syphilitic treatment, followed by a second Wassermann test. If this routine is followed a number of unnecessary operations will be avoided, and the patients saved a great deal of unnecessary crippling in after life.

Orthopedic Treatment.—In the majority of syphilitic bone and joint lesions local orthopedic treatment is unnecessary. If the joint is very painful, or if bone destruction has taken place, temporary immobilization is desirable. This may be secured by braces or a plaster-of-paris bandage. These should be removed as soon as the symptoms improve. In syphilitic arthritis the object is to restore function and for that reason immobilization should be maintained only as long as is necessary to relieve the acute symptoms and to protect the joint.

Medical Treatment.—The medical treatment for bone and joint syphilis is the same as that employed in the treatment of all other types of syphilis. In children arsphenamine and neoarsphenamin give very excellent results. These may be combined with mercury inunctions and the administration of Gray powder. In older children and adults the administration of bichloride of mercury and potassium iodide by mouth seems to have a very beneficial results in relieving the symptoms and in securing rapid and marked improvement in the lesion. This should be combined with arsphenamine. Joint symptoms do not seem to improve as rapidly after the administration of arsphenamine as with the use of bichloride and potassium iodide. Nor does the improvement seem to be as rapid or as marked after the administration of arsphenamine followed by a course of mercury injections. In older cases the use of potassium iodide seems to be essential. In younger children Gray powder gives very good results in relieving the symptoms and in curing the lesion.

The fact must be constantly borne in mind that bone and joint syphilis is only a manifestation of a general syphilitic infection. These conditions yield very rapidly to treatment, but the disease is not cured with the disappearance of the symptoms. The patient must be given the same exact and rigid treatment as he would be given for any other type of syphilis.

Recapitulation.—Syphilis of the bones and joints is much more common than has been supposed. The more general use of the Wassermann test and the *x*-ray shows that a large number of cases supposed to have been tuberculosis, osteomyelitis, infectious arthritis, synovitis, etc. is really syphilitic in origin. A Wassermann test should be made, as a routine, in all cases of bone and joint disease. When the Wassermann test is positive the diagnosis should be confirmed by the therapeutic test. The fact must not be overlooked, however, that the Wassermann is often negative, even when syphilis is present, especially in congenital syphilis. All cases in which there is a history of indefinite joint tenderness, multiple joint involvement, pain, whether increased at night or not, or where the pain or functional disability is not proportionate to the amount of joint involvement, should be regarded with suspicion. In these cases a very careful history should be made, to elicit any suggestion of a previous venereal infection, either in the patient or in the parents. In this connection the fact should be emphasized that frequently a syphilitic infection may not be recognized by the patient. In children a diagnosis may often be made by noting other signs of congenital syphilis.

The *x*-ray is of great value in making a diagnosis. In the majority of cases it will show the typical lesions of bone syphilis. The joint lesion is usually secondary to the bone lesion. In some cases the bone or joint picture may closely simulate other forms of disease; in these cases *x*-rays of some of the other bones will generally show a typical periostitis. In some cases where the lesion is primary in the synovial membrane the *x*-ray picture may be so similar to other lesions that a diagnosis from the *x*-ray is impossible.

The symptoms of bone and joint syphilis are so varied that they may simulate almost all types of bone and joint infection.

It is the author's belief that in all cases where there is any reasonable doubt as to the diagnosis, syphilis should be suspected, and every possible means should be employed to establish the diagnosis. The absolute necessity of this is obvious, as the difference between the treatment of syphilis and the other joint conditions is so radical, that the prognosis depends essentially on the correctness of the diagnosis and the resulting treatment.

The treatment of bone and joint syphilis is directed primarily at the constitutional infection, and should consist of the most thorough and approved methods. The bone and joint symptoms rapidly yield to treatment but the patient must not be considered cured as soon as the local lesions are apparently well.

Orthopedic treatment is symptomatic. When there is much pain on function or when much joint destruction has taken place, rest of the diseased joint is indicated. This can best be secured by some form of support, either a brace or plaster-of-paris cast. In the ordinary case support is usually not necessary, as the symptoms generally improve rapidly under constitutional treatment.

The author wishes again, and finally, to emphasize the importance of constantly considering syphilis as an etiological factor in bone and joint lesions. The importance of syphilis in these conditions is not, as yet, fully realized. Much time has been wasted and much damage done by not thinking of syphilis in making a diagnosis of bone and joint lesions. This is not only the belief of the author, but the belief of most other writers on the subject in recent years.

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CHAPTER CXII

TRAUMATIC DISLOCATIONS

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Definitions.—A dislocation is a permanent abnormal displacement of the articular surfaces of the bones entering into the formation of a joint.

A traumatic dislocation is one which is produced suddenly by external violence or muscular action or a combination of both of these forces. The external violence may be direct, *i.e.*, near the end of a bone, or indirect, *i.e.*, at a distance from the end of a bone.

This chapter is a consideration of traumatic dislocations only. It does not deal with other types of dislocations (spontaneous, congenital etc.) in which the displacement is occasioned by malformation of a joint or pathological changes in or about a joint.

A *complete* dislocation is one in which the articular surfaces are no longer in contact or in which they touch only by their edges. An *incomplete* dislocation (or subluxation) is one in which the articular surfaces are dislocated to a lesser degree.

A dislocation is said to be *compound* when the joint cavity is open to the outer air. A dislocation is said to be complicated when lesions of other structures in or about the joint exist, such as fracture or laceration of vessels or nerves.

Multiple dislocations are those in which two or more bones are simultaneously dislocated. When both ends of a bone are dislocated, the dislocation is said to be *double* or *total*. When symmetrical dislocation on opposite sides of the body occurs, as of both shoulders or both hips, the dislocation is said to be *bilateral.*

A uniform nomenclature of dislocations has not been established. As a rule the bone more distant from the mid-line of the body is said to be dislocated, *e.g.*, dislocations of the hip or of the shoulder are spoken of as dislocations of the femur or of the humerus. As an exception to this rule, however, it is customary to speak of dislocation of the outer end of the clavicle rather than dislocation of the acromion. Certain qualifying terms indicating the direction of the dislocation are also in common use, *e.g.*, inwards, outwards, forwards, backwards. The direction of the dislocation may also be indicated by the relation which the displaced bone bears to some anatomical part, e.g., subacromial, subglenoid, infraspinous, etc.

Signs and Symptoms.—A history of injury is elicited. Occasionally an accurate description of the injury may reveal the mechanism of the dislocation. Pain is present but its intensity varies within wide limits according to the character of the injury and the temperament of the individual. Swelling is usually present. It may be slight in amount or so extensive as to obscure bony landmarks. The old teaching that fractures result in abnormal mobility and dislocations in loss of mobility has numerous exceptions. In dislocations, if the joint capsule and peri-articular ligaments are badly torn, it is obvious that mobility of the joint may be excessive. In fractures of subperiosteal or impacted type it is obvious that no abnormal mobility at the site of fracture exists and muscular spasm may lessen the degree of motion at the nearest joint. As a rule, however, in dislocations there is limitation of motion both from muscle spasm and mechanical obstruction. There is always some degree of hemorrhage. It may be extremely slight or so marked as to distend the joint capsule or cause extensive extravasation into the soft parts. Rarely in children are large vessels torn which jeopardize the viability of the extremities. Circulation of parts distal to the injury may not infrequently be deranged by pressure upon important vessels. There may be temporary loss of sensation or motion from stretching or concussion of peripheral nerves. In childhood severance of nerves as a complication of dislocations is unusual. In dislocations of the vertebræ the spinal nerves and cord are exposed to the same dangers as in adults.

Examination of Patient.—There are few instances which more clearly demonstrate the surgeon's familiarity with the handling of children than his examination for fracture or dislocation. The custom of sending the patient with suspected fracture or dislocation immediately to the roentgenologist without examination is deplorable. This custom does not indicate lack of time but rather lack of patience, lack of knowledge of the handling of children, and lack of a desire to educate the tactile sense. As soon as the confidence of a child has been gained, it is usually possible to obtain from the examination all the data necessary for a diagnosis. There are many ways of gaining the child's confidence but above all are calmness and gentleness of manner. A very successful method is to lead the child to believe that the examination is a kind of game which he and you are playing. Much can be learned by careful inspection without touching the child. In case of an injury to the elbow, for example, the patient sits opposite you in the mother's lap. Both arms are viewed from the front, noting the amount of swelling, its localization, the prominence or obscurity of bony landmarks, the carrying angle, etc. The mother then turns upon the stool so that the uninjured and then the injured arm may be viewed from the side. The mother then turns appropriately so that both arms may be viewed on their inner and posterior aspects. The bony prominences of the *uninjured* elbow are then palpated and the movements of flexion, extension and rotation passively performed. A tentative diagnosis has probably been made from inspection, and therefor when the affected elbow is palpated and passively moved, the points which may be expected to be tender and the motions which may be expected to be painful are reserved till the close of the examination. Prior to this the color and warmth of the hand have been observed, the radial and ulnar pulses tested, and movements of the fingers, thumb and wrist actively executed in imitation of the surgeon (as part of the "game") to determine the integrity of the median, ulnar and musculo-spiral nerves. The more frequently that children are examined in this calm, gentle, deliberate yet playful manner, the more rapidly can the examination be performed and the more readily may abnormal conditions be correctly diagnosed. The *x*-ray is relied upon then to confirm the diagnosis and to reveal the accuracy of the reduction. Although the surgeon may feel positive of his diagnosis from his examination, nevertheless it is important in children to have radiographs made in order to determine the integrity of the epiphyses before reduction is attempted.

Treatment.—A general anesthetic is usually advisable. Its use is not only more humane but it overcomes muscle spasm. In consequence the manipulation of reduction requires much less force and is accomplished without further damage to the soft parts. Brutal forcible manipulation with or without an anesthetic may accomplish the reduction but may leave the soft parts pitifully and perhaps seriously lacerated. No reduction should be attempted without having previously pictured the mechanism of its occurrence. We should then attempt with as much gentleness as possible to execute maneuvers which retrace the steps of this mechanism. The observance of this principle will facilitate reduction and consequently obviate needless mutilation of the soft parts. In fact the retracing of the mechanical steps of the dislocation may be the only way in which a bone-end may be successfully replaced through a rent in the capsule. Brutal unintelligent efforts to obtain a reposition of bone-ends may be absolutely futile on account of the interposition of capsule or other soft parts. After reduction, appropriate measures for immobilization of the joint must be instituted for a period, long or short, according to the site and character of the dislocation. In children joint ankylosis is not the bête-noir it is in adults. Perhaps therefore we are safer in erring to the side of immobilizing a little too long. Whether motions be resumed early or late, it is important to restrict that position which favors a recurrence of the displacement. I cannot conclude this paragraph more fittingly than by quoting the words of Stimson:[1] "The easy reduction of most dislocations under ether by direct pressure in suitable directions upon the projecting ends of the bones is an indication that ligamentous obstacles of importance do not exist and that the chief opposition is furnished by the muscles spasmodically contracted on all sides of the joint, and the inference is too often drawn that, provided this opposition is overcome by force or by anesthesia, the surgeon need not particularly concern himself with the attitude of the limb during his efforts to reduce. But the success of a faulty method should not make us unmindful of its defects; our work should be done skillfully as well as successfully, and even if our errors will pass undetected and their consequences be promptly repaired, we should not lightly commit them."

DISLOCATIONS OF THE LOWER MAXILLA

Dislocations of the lower jaw are unusual in childhood. They may be occasioned by external violence or by muscular action. The displacement is usually forwards and bilateral. Unilateral displacements backwards, outwards, or upwards are very rare, and occurring by external violence, are associated with fractures of the mandible, the external auditory canal, or roof of the glenoid cavity.

Forward Dislocations.—These are usually bilateral. They may be caused by external violence, such as falls, blows, or rarely at delivery, or by muscular action, such as in screaming or in attempting to put unduly large objects into the mouth. Various views have been advanced in explanation of the mechanism of this dislocation. It seems quite likely that when the jaw is opened too widely the condyle slips along the eminentia articularis, stretching the external ligament. When it has passed the forward border of the articular surface, the mouth closes slightly, the ligament contracts and prevents the condyle from resuming its normal position. Rarely the meniscus of the condyle may be detached and prevent reduction but usually the obstacle to reduction is the contracted lateral ligament. Therefore the rational procedure for reduction is to stretch the lateral ligament by depressing the chin (i.e., opening the mouth a little wider) and then pushing the jaw backwards. It is interesting that this is the procedure which is advocated by Hippocrates[2] in his treatise on articulations.

Symptoms.—The jaw is thrust somewhat forwards, the mouth almost always open and drooling, the cheeks flattened, mastication impossible and swallowing difficult. The empty glenoid cavities leave palpable depressions in front of the ears.

Prognosis.—The prognosis for reduction and subsequent use of the jaw is good. Unfortunately there is a tendency to recurrence. This may be because there is so little inconvenience after reduction that use of the jaws may be too early resumed.

Treatment.—The patient's head is held firmly by an assistant. The operator's thumbs are placed within the mouth, the fingers along the lower jaw. The chin is depressed by the operator's thenar eminences and the jaw is then lifted backwards by the fingers and thumbs. As explained above, the initial depression of the chin (i.e., opening the mouth still further) again places the contracted lateral ligament on the stretch and permits the condyle to slip backwards into the glenoid cavity. In view of the probable mechanism of the dislocation, this procedure seems more rational than simple depression of the jaw at the molar teeth followed by a backward thrust. Anesthesia may be necessary to overcome muscular resistance. The mouth should be kept closed for two or three weeks by a simple bandage. A four-tailed bandage to the chin is satisfactory. The diet must necessarily be of liquids and soft solids. Rarely is open reduction necessary.

DISLOCATION OF THE STERNUM

Under this heading are included dislocations of the manubrium upon the gladiolus, of the gladiolus upon the manubrium, or of the ensiform upon the gladiolus. All are extremely rare. The most frequent seems to be displacement of the second piece forwards and upwards on the first piece. External violence or muscular action may produce the lesion. External violence may be in the nature of falls upon the chest or blows from moving objects. Muscular action may be in the nature of tetanic convulsions, falls upon the back with hyperextension of the spine, etc. Ancelet (quoted by Stimpson)[3] records a boy 13 years old who, while exercising on parallel bars with his chest bent forwards, suddenly touched the floor with his feet. The left upper corner of the gladiolus was displaced forwards on the manubrium. Guines[4] records a boy 13 years old who during a tetanic convulsion had his gladiolus displaced 3 in. forwards on the manubrium. The deformity persisted after his recovery from the tetanus on the eighteenth day.

Symptoms.—In case of associated injury of the spine or thoracic viscera, the symptoms of dislocation may be of minor importance. In any case the deformity must be readily detected. By noticing the relationship of the displaced region to the costal cartilages of the second ribs, the condition should be differentiated readily from fracture.

Prognosis.—The prognosis is usually bad on account of associated spinal or thoracic lesions. In cases due to lesser violence, the prognosis is good. Cases with unreduced dislocations of the sternum have lived without great inconvenience for years.

Treatment.—The rational treatment in dislocation of the sternum forwards on the manubrium would be hyper-extension of the dorsal and cervical spine with inward pressure over the gladiolus followed by a tight swathe or adhesive strapping. If simple measures of this nature fail, operative interference should not be undertaken unless grave symptoms due to compression of thoracic viscera are present.

Dislocation of the Ensiform Process.—Dislocations of the ensiform process on the gladiolus are so rare in adults as to be surgical curiosities, and perhaps never occur in children. Localized pain and distress and vomiting after eating have been described. Symptoms have disappeared spontaneously or have been relieved by digital manipulation or open reduction.

DISLOCATIONS OF THE CLAVICLE

The clavicle may be dislocated at either end or at both ends simultaneously. Dislocation of the acromial articulation is the most common.

Dislocations of the Sternal End of the Clavicle.—This is an unusual injury in children. The displacement may be forwards, backwards, or upwards.

Forward Dislocation.—This may be produced by muscular action or direct violence. The usual mechanism is as follows: A backward or backward and downward force upon the shoulder stretches the anterior sternoclavicular ligament. Continuation of the force pries the under surface of the clavicle against the first rib. The anterior ligament (and perhaps others) then rupture. Part of the articular surface may remain in contact with the sternum (incomplete type), or the articular surface may be entirely separated (complete type). In the latter the end of the clavicle is not infrequently drawn down so that it comes to lie upon the anterior surface of the sternum. The displacement has occurred at delivery, and in a babe only 10 months old by a fall from bed (Wright[5]).

Diagnosis.—Diagnosis is readily made in the complete type. In the incomplete type the abnormal anterior prominence of the sternal end of the clavicle is obvious. The restoration of the contour of the parts by a simple backward thrust on this prominence establishes the diagnosis. A small scale or chip of bone may be torn away and an unrecognized but gross fracture (unusual in this situation) should be readily appreciated.

Prognosis.—Prognosis for restoration of function is good. The prognosis for complete reduction is only fair. A tendency to habitual or recurrent dislocation may result.

Treatment.—Reduction is usually simple. In the incomplete types simple backward pressure, in the complete type elevation of the arm, lifting the sternal end to its proper level and then pressing backwards and bringing the shoulder forwards. The maintenance of the reduction is difficult. A figure of eight bandage about both shoulders drawing them forwards and crossing in front of the chest combined with a sling to carry the weight of the arm may suffice. A thick soft pad held by some moldable splint (felt, leather or plaster) over the displaced bone with rest in bed in recumbent posture may be necessary. The danger of pressure slough is to be remembered. Treatment should be carried out for two or three weeks but free movement of the arm should not be permitted for a couple of weeks more.

In habitual or recurrent dislocation Stimson[6] has successfully used peri-articular injections of alcohol, few drops of alcohol injected in front of and below the joint with the arm immobilized. Several injections at weekly intervals may be necessary.

Dislocations of the Acromial End of the Clavicle.—The outer end of the clavicle may be displaced upwards, or downwards and backwards, or downwards and forwards beneath the coracoid. The first is by far the most common and the last extremely rare.

Upward Dislocations (**Supra-acromial**).—This type of dislocation may be caused by direct violence or muscular action. The mechanism in the former case is usually a downward blow on the acromion associated with a contraction of the trapezius. It is to be remembered that the upper surface of the outer end of the clavicle is normally higher than that of the acromion and varies in different individuals. It is also to be remembered that the point of the shoulder is formed not by the outer end of the clavicle but by the outer border of the acromion process, a fact which is forgotten with surprising frequency. The articulation lies on the inner border of the acromion. It is well in all injuries in this region to trace the spine of the scapula upwards and forwards to the acromion, to palpate the outer and anterior margins of the acromion and then to trace the border of the clavicle outwards to its acromial end. There may be present only localized tenderness, pain on movements of the arm, and an increased prominence of the end of the clavicle above the acromion which is readily reduced by downward pressure but usually immediately recurs when the pressure is relieved. This is the so-called incomplete type. In the complete type the end of the clavicle is displaced upwards its entire thickness and may override the acromion either forwards or backwards.

Prognosis.—In the incomplete type, although reduction may not be absolute, the prognosis for function is good and the chance for deformity slight. In the complete forms, reduction is most difficult to maintain and may not be accomplished at all. The loss of function is not always proportional to the degree of persistent displacement, some patients with marked persistent displacement may experience no disability, and vice versa.

Treatment.—The difficulty is more often the maintenance of reduction rather than the reduction itself. This is due mostly to the weight of the arm and perhaps in part to spasm of the trapezius. A simple retention device which is as effective as any is the Stimpson (sometimes called the Jones) strapping. The arm is held at the side with elbow bent at right angle while an assistant retains the reduction by downward pressure on the clavicle. The middle of a strip of adhesive plaster more than three times the length of the upper arm is then placed under the elbow, one strip then carried upwards along one side of the arm, across the acromio-clavicular joint and downward onto the back, the other carried up the other side of the arm across the acromio-clavicular joint and downwards onto the chest. The forearm may be held in a simple cravat sling, preferably with the arm at a right angle. This device is to be used for several weeks. Care is necessary to avoid pressure sores over the articulation. Operative interference is occasionally necessary for unsightly deformity or disability. Periosteal and ligamentous sutures should be sufficient. In incomplete cases, simple downward pressure on the outer end of the clavicle accomplishes the reduction. In complete cases with overriding of the clavicle on the acromion forwards or backwards, it is necessary to draw the arm outwards, either forwards or backwards and then lift it upwards to elevate the acromion and at the same time push downwards on the outer end of the clavicle. In a child this may readily be done by allowing the arm to hang at the side of the chest; by introducing both hands into the axilla, one from in front and one from behind, so that the backs of the fingers rest against the chest wall. The forefingers are now at the top of the axilla. The thumbs are carried over the shoulder, front and back, to rest upon the outer end of the clavicle. The upper part of the humerus is now grasped in the palms, drawn outwards, forwards or backwards, and then upwards while the thumbs depress the clavicle.

DISLOCATIONS OF THE VERTEBRÆ

In order to understand the dislocations which may occur between vertebræ, it is necessary to examine the skeleton or still better a specially prepared specimen of the spinal column with the ligaments in place. It is necessary to understand the normal curves of the spine, the relative size and shape of the bodies of the vertebræ and their discs, the shape and inclination of the articular processes. It will be appreciated that in undue flexion or extension of the spine, if the articulation between the bodies of two vertebræ gives sufficiently, there may follow dislocation of the superior or inferior articular process. No rotation would be concerned in the mechanism of such a displacement, merely hyper-flexion or hyper-extension. Both inferior articular processes of one vertebra may be displaced over and in front of the superior articular process of the vertebra below, so that they come to lie in the intervertebral notches of the lower vertebra. This is *bilateral forward* dislocation by flexion.

If the dislocating force be one of rotation or abduction rather than of flexion or extension, then there may be displacement of the articular process of one side only. This is *unilateral* dislocation by rotation or abduction. If the rotary force be still greater, there may be an associated backward dislocation of the articular process on the opposite side. This is *bilateral* dislocation *in opposite directions.*

Again it may be possible by localized external violence to displace one vertebra upon another laterally. From the inclination of the articular processes of the cervical and upper thoracic regions such a dislocation is seen to be possible. This is *transverse* dislocation. Bell reported such a dislocation in a child who had been run over by a coach.

To recapitulate:

By hyper-flexion or hyper-extension there may be produced
  bilateral forward dislocation
  bilateral backward dislocation.

By hyper-rotation or hyper-abduction there may be produced
  unilateral forward dislocation
  unilateral backward dislocation
  bilateral dislocation in opposite directions.
By localized external violence there may be produced
  transverse dislocation.

Complications.—Inasmuch as dislocations of articulations other than those of the spine may be attended by fracture, avulsion of bony prominences, or epiphyseal separation, just so may spinal disarticulations be attended by such osseous injury. In fact dislocations of vertebræ are not infrequently so complicated.

The spinal cord may or may not be injured. If injured, it may be in any degree from slight contusion to complete crush. The violence of the dislocating force, the size of the bony canal, and the strength of the intervertebral ligaments govern the degree of damage to the cord. However it is not exempt from injury at any level, but apparently less liable to injury in certain types of dislocation. It is least apt to be affected in bilateral dislocation in opposite directions, more often in unilateral dislocation forward or backward, and usually in bilateral dislocation forward or backward. Paralysis is usually bilateral, its nature and extent depending upon the level and severity of the cord injury. Compression of the cord may be due to hemorrhage rather than to bone and this may occur within or without the cord. There may be motor, sensory, or vasomotor disturbances. There may be fecal and urinary incontinence, urinary retention, priapism.

Spinal nerves may escape injury, may be stretched, completely avulsed, or compressed at the intervertebral foramina.

Rupture of blood-vessels always occurs in vertebral dislocations. The hemorrhage may be slight or sufficiently profuse to produce compression of the cord.

Diagnosis.—In cases which are *not* seen immediately after the accident, something may be learned by inspection, palpation, and tests of motor and sensory function. In cases seen immediately after the accident, we are greatly handicapped because it is known that even slight turning of the trunk or neck may cause irreparable damage to the cord or nerves or even sudden death. Not infrequently a recently injured child is seen for the first time lying upon his back. Before moving him it is best to note his color, respiration, attitude of his head and limbs, and to secure a history of the accident. Still without moving him it is best to loosen his clothing only sufficiently to permit observation of reflexes, cutaneous sensation, motion of limbs, priapism. Palpation of the posterior wall of the pharynx and of the abdomen, if the child be thin, may give information regarding the position of the vertebral bodies. If there is no evidence of paralysis, movements in undressing him should nevertheless be very cautious, and it is best that he be carefully transferred to a Bradford frame. Upon the

Bradford frame he may be taken to the *x*-ray room, or better, if the hospital has a portable *x*-ray apparatus, it may be taken to him. Anteroposterior views may be taken by simply slipping the plates under him. Obliquely lateral views may be taken if the frame is lifted somewhat from the bed.

In cases which are not seen immediately after the accident, perhaps the back may be safely inspected and palpated. Localized rigidity may be present and in addition a prominence, depression, deflexion, or tenderness of a spinous process or similar abnormalities of one or both transverse processes of a vertebra may be noted. From such data, mindful of the possible types of dislocation, a conjecture may be made regarding the character of the injury in the case at hand.

Treatment.—In cases with paralysis which are seen immediately after the accident, prompt reduction should be attempted and the parents or some responsible person should be acquainted with the risks of the procedure. In cases with paralysis which has occurred at the time of the accident, there is considerable doubt as to the benefit to be drived from reduction. In such cases it is often impossible to determine whether the paralysis is due to severe contusion of the cord or to laceration of the cord. Despite this uncertainty, it seems justifiable cautiously to attempt reduction. Immediate laminectomy in these cases does not seem warranted; for, if the paralysis is due to simple contusion of the cord, restoration of function may occur without laminectomy, whereas if the paralysis is due to laceration of the cord, laminectomy accomplishes nothing.

In cases with paralysis which are not seen immediately after the accident but in which the paralysis has been of more or less gradual development, reduction is unquestionably indicated. The gradual onset of paralysis may be interpreted as compression of the cord from hemorrhage. After reduction if the paralysis does not improve, laminectomy is justifiable.

In general, anesthesia should be employed. If the dislocation is comparatively slight, moderate pressure may effect reduction, as in a case related to Blasius by Richter.[8] Richter attempted to reduce a slight dislocation of the third cervical vertebra in a boy of 11 or 12 by traction on the head. This intelligent lad understood what the surgeon had tried to do and on the way home he leaned his head and shoulders against a wall and pressed with his thumb on the convex side of his neck. The following day Richter found the neck straight, without spasm and freely movable. The special procedures for dislocations of the various levels of the spinal column are noted below. After reduction it is well to prevent recurrence by a plaster jacket or collar. In cases in which retention for some time is necessary a leather jacket or collar reinforced with metal ribs may later be substituted. In cases in which reduction cannot be accomplished, such retention apparatus should always be applied to favor fibrous or bony fixation. In paralyzed cases attention to the excretory demands of the patient and attention to bed sores require skilled nursing. A Bradford frame is of great assistance. Suprapubic cystostomy may be preferable to repeated catheterization or an inlying catheter.

Dislocations of the Cervical Vertebræ.—The cervical vertebræ, especially the fifth, are more prone to dislocation than those of the thoracic or lumbar region.

Dislocation of the Atlas (on the Occiput).—Dislocations of the atlas are rare, as might be expected from its sheltered position under the base of the skull, its deep cup-shaped articulations with the occiput, the strong ligaments of this articulation and the check ligaments from the odontoid process. Displacement is from direct violence, a blow on the back of the neck as in Costes'[9] case. This boy was beaten on the back of the neck leaving the head turned to one side with the chin almost resting upon the chest. Paralysis ensued and he died five months later. There was found a fracture of the odontoid process and bilateral dislocation forwards of the atlas, complete on the left, partial on the right. Most cases, however, die instantly. Mixter and Osgood[10] report a brilliant case in a boy of 15 who had fracture of the odontoid and anterior dislocation of the atlas. Nine months after injury an open reduction was done and maintained by tying together the posterior arch of the atlas and the spinous process of the axis with a stout silk ligature. A previously made, perfectly fitting leather cuirass was worn for two months. Two years later the boy was in excellent condition and had led an active life.

Treatment.—Reduction should be accomplished by steady traction on the head and such pressure on the vertebræ as the nature of the displacement indicates, followed by retention in plaster-of-paris collar. Subsequently a leather collar reinforced with metal ribs may be substituted.

Dislocations of the Axis.—This is more frequent than dislocations of the atlas on the occiput. The check ligaments may be ruptured permitting the odontoid process to pass beneath the transverse ligament, but more often the transverse ligament or odontoid process are ruptured. Sedillot[11] reports a girl whose head was forcibly twisted from behind by a man. The head remained deviated to the left with the chin directed downward. Paralysis ensued and death occurred seven weeks later. This has been considered a bilateral dislocation in opposite directions.

Bilateral backward or forward dislocation may be caused by a fall or blow on the head especially one which flexes the head onto the chest. The cord is usually, but not necessarily, injured. There is usually pain, rigidity of the neck, fixing of the head either with the chin depressed in the median line or to one side. The prognosis is almost always bad. The risk of reduction is great but seems justifiable. Upward traction should be made upon the head with or without pressure on the back of the neck as the conditions of the case indicate. Any attempt at reduction should be brief and deliberate followed immediately by a plaster-of-paris collar. An appropriate leather collar reinforced by metal ribs may be substituted after several weeks.

Dislocations of the Lower Six Cervical Vertebræ.—The lower six cervical verebræ are the most frequently dislocated vertebræ, especially the fifth. The most common types are bilateral forwards (dislocations by hyper-flexion) and unilateral forwards (dislocation by hyper-rotation and hyper-abduction). The causes may be either external violence or muscular action.

Diagnosis.—Bilateral dislocations forwards. The neck is usually held rigidly. The face is commonly directed downwards, there is no lateral angulation of the neck. Anteroposterior angulation may be marked. Distinct depression may be felt in the mid-line posteriorly, due to the absence of the spinous process from forward dislocation of the vertebræ. If not beyond reach, a prominence due to the body of the vertebra may be palpated in the pharynx.

Unilateral dislocations forwards. The face is usually turned to the side opposite that on which the dislocation has occurred. There may be a distinct lateral angulation of the neck on the dislocated side. The spinous process may be felt deflected to the same side and perhaps the vertebral body may be felt unduly prominent in the pharynx. Paralysis may or may not be present. (See general considerations on dislocations of vertebræ above.)

Prognosis.—In cases without paralysis in which reduction is accomplished and immobilization maintained through a sufficiently long period to insure firm fibrous or bony repair, the prognosis is favorable. In cases with paralysis, the reader is referred above to general considerations on dislocations of the vertebræ. As is to be expected, the prognosis is more favorable in unilateral than in bilateral dislocations forward.

Treatment.—In cases of unilateral dislocations forward or bilateral dislocations in opposite directions (especially those due to violent muscular action) the reducton may usually be simply and safely performed. Usually in unilateral forward dislocations, the face is turned and the neck is bent to the side opposite that on which the dislocation is present. The rational procedure is first to bend the head further in the same direction to disengage the articular process and then exert traction upwards on the head, at the same time turning the face to the mid-line and tipping the head towards the dislocated side.

In bilateral dislocations forward reduction has been accomplished by traction upwards on the head combined with backward pressure upon the column below the dislocation.

After reduction the neck should be held in retentative apparatus of plaster of paris which prevents both flexion and rotation.

DISLOCATIONS OF THE SHOULDER

Although traumatic dislocations of the shoulder in adults are about as numerous as dislocations of all the other joints combined, they are rare in children. In fact in Kronlein's analysis of 400 recent traumatic dislocations[12] there were only two of the shoulder in children. Fractures of the clavicle in children may be the equivalent of dislocations of the shoulder by direct violence in adults, and dislocations of the elbow in children may be the equivalent of dislocations of the shoulder by indirect violence in adults. It is to be remembered also in children that separation of the upper humeral epiphysis occurs more frequently than dislocations of the joint and no injury of the shoulder should be treated without knowledge of the condition of the epiphysis. It is true that so-called congenital dislocations and those consequent upon obstetrical paralysis are not infrequent but these are not within the province of this chapter.

The anterior or subcoracoid dislocations are the commonest and these alone will be considered in detail. All other forms are rare and will merely be enumerated: *Viz.*, the extreme anterior dislocation (intracoracoid, subclavicular); downward dislocations (subglenoid and luxatio erecta); posterior dislocations (subacromial and subspinous); and upward dislocations (supraglenoid).

Subcoracoid Dislocations.—As the name implies, the head of the humerus comes to lie in a position between the anterior margin of the glenoid fossa and the coracoid process. This may be produced by direct or indirect violence or by muscular action. The commonest mechanism is by hyper-abduction. The ascent of the humerus is arrested by the outer edge of the acromion, directing the head downwards and forwards against the anterior and lower part of the capsule. Continued elevation tears the capsule in this location, and the head of the bone may or may not be forced through. If the head is forced through, contraction of the inward rotators draws it still further forwards and inwards past the anterior edge of the glenoid cavity. Upon descent of the arm, the head is forced upwards along the inner side of the joint. It is to be noted that the outer and posterior parts of the capsule remain intact; for the rational method of reduction by manipulaton depends upon this fact. Injuries of the circumflex nerve and circumflex and subscapular arteries are possible but not likely in children.

Diagnosis.—The trunk is inclined towards the injured side. The forearm of the injured side is held in the other hand to support the weight of the arm. The rounded prominence of the deltoid is more or less flattened and palpation in this region fails to detect the head of the bone. A prominence may be seen on the anterior wall of the chest between the glenoid cavity and the coracoid process. This feels hard and globular and will roll under the fingers if the arm be gently rotated. This is the head of the humerus and its recognition establishes the diagnosis. It may also be noted that the elbow of the affected side is held at a little distance from the trunk and that efforts to adduct the arm are resisted and painful. The hand of the affected side cannot be placed upon the opposite shoulder.

Treatment.—The rational maneuvers for reduction are those advocated by Kocher[13] because they follow in reverse order the mechanism of the dislocation. This method may be divided into three steps. (1) The elbow is brought to the side of the trunk with the forearm at right angles to the upper arm. By grasping the forearm, the humerus is forcibly rotated outwards, still holding the elbow firmly to the side. This maneuver will untwist the capsule, distend its intact outer and posterior portions and open wide the rent in its anterior and inner portion. The head is still outside the glenoid cavity. (2) With the humerus still forcibly rotated outwards and the forearm still at right angles to the upper arm, the elbow is lifted forwards and inwards until the upper arm is nearly at right angles to the trunk. The head of the humerus should now be within the lips of the ruptured capsule on the edge of the glenoid cavity. Sometimes this maneuver carries the head through the rent onto the glenoid cavity and completes the reduction. (3) The humerus is sudenly rotated inwards, i.e., the forearm which is still flexed at a right angle to the upper arm is suddenly carried to the opposite shoulder. This maneuver turns the head through the rent and into the glenoid cavity. This method will usually succeed and is to be tried first. Should it fail, traction may be made downwards and somewhat outwards upon the arm combined with direct pressure on the head and slight internal rotation. Direct

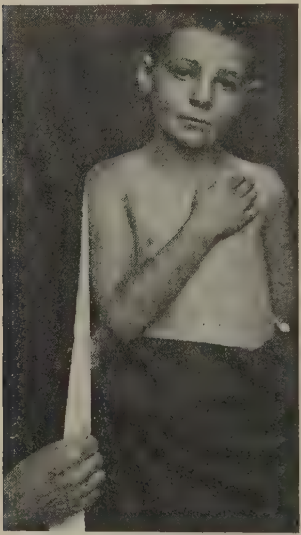

Fig. 148.—Adhesive strap carried from back over tip of shoulder and down postero-lateral aspect of arm to elbow.

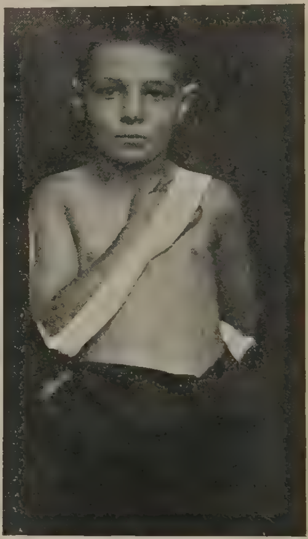

Fig. 149.—Adhesive strap carried beneath elbow, below posterior aspect of forearm and hand, and over opposite shoulder to the back.

traction downward on the arm with the heel in the axilla may be effective but is risky.

After reduction the arm should be immobilized for two or three weeks. This may be accomplished by a Velpeau bandage or by a long strip of adhesive plaster about 2½ in. wide which starts over the back of the affected shoulder, passes down the outer side of the upper arm, under the elbow, along the forearm and dorsum of the hand as they lie against the chest wall, to the back of the opposite shoulder (Figs. 148, 149). The axilla, bend of elbow and anterior surface of the chest should be well powdered. A sling should be worn for another two or three weeks. Passive movements may be started during this period but external rotation and abduction are to be avoided.

DISLOCATIONS OF THE ELBOW

Traumatic dislocations in children occur most frequently at the elbow. Both bones may be dislocated backwards, forwards, inwards, outwards, or in divergent directions, or the radius or ulna alone may be dislocated. The commonest forms are backward dislocation of both bones and dislocation of the radius downwards (by elongation). Krönlein[14] in his analysis of 400 recent traumatic dislocations lists 109 of the elbow. Ninety-four were of both bones backwards and of these 66 occurred in the first two decades of life. Fifteen were of the radius alone, and 14 of these occurred in the first two decades of life.

Dislocations of Both Bones of the Forearm Backwards.—This is the commonest dislocation in children and has been regarded as equivalent to dislocations of the shoulder in adults by indirect violence. It probably is always caused by a fall upon the palm of the outstretched hand, i.e., with the elbow in complete extension. The internal lateral and anterior ligaments are always torn, the external lateral usually, and the orbicular rarely. The internal epicondyle may be avulsed and carried upwards and backwards with the lateral ligament. The periosteum on the posterior surface of the humerus may be stripped up by the head of the radius and in such cases is responsible for the subsequent bony thickening in this region, especially in the unreduced cases (Stimson,[15] Cotton [16]). The external epicondyle may be separated or the coronoid process may be fractured. In certain cases there may be separation of the upper radial epiphysis or of the olecranon epiphysis; the former by being jammed by the descending lower extremity of the humerus, the latter by the forcible impingement against the back surface of the humerus. The flexor muscles of the hand are sometimes torn off at their origin, the brachialis anticus sometimes at its insertion, and the biceps tendon may be displaced outwards and caught in back of the external epicondyle. Large blood-vessels are rarely injured. The ulnar nerve may be stretched or torn, especially in those cases where there is separation of the internal epicondyle. The median and musculospiral nerves are occasionally injured.

Diagnosis.—Inspection shows the forearm apparently shortened and a prominence on the posterior surface of the arm in the region of the elbow which is exaggerated by flexion. In other words the anterior posterior diameter of the joint seems increased whereas the transverse diameter seems normal. The forearm is held usually in a position midway between right angle flexion and complete extension. The carrying angle may not be disturbed or there may be cubitus varus or valgus, according to the extent of the laceration of the ligaments. Palpation demonstrates the abnormal relations of the olecranon and radial head to the internal and external epicondyles. The child should be asked to extend and flex the wrist and fingers and to adduct and abduct the fingers in order to determine the integrity of the median, musculo-spiral and ulnar nerves. Pulsation of both radial and ulnar arteries at the wrist should be tested. In no case should reduction be attempted unless the abnormal relation of the bony prominences above mentioned is established, and in all cases in which it is readily possible, an *x*-ray should be taken, not especially to confirm the relation of these bony prominences but rather to determine the integrity of the epiphyses.

Prognosis.—In recent uncomplicated cases the prognosis is good. In cases seen after a few weeks, the prognosis for closed reduction is fair. If moderate force fails, open reduction is indicated. The functional result after operation depends upon the amount of bony proliferation at the back of the humerus and in the sigmoid cavity.

Treatment.—The methods of reduction are numerous. All of those which depend upon force rather than upon a reversal of the mechanism of production are to be condemned. Such methods are forcible flexion over the operator's knee, about a post, or over a bar. These methods succeed only by increasing the laceration of the ligaments. They may even be attended by fractures or epiphyseal separations (especially the olecranon). The rational method is as follows: (1) Extension, or even hyper-extension, of the forearm. (2) Downward and forward traction on the forearm (or forward pressure on the olecranon and head of the radius) combined with counter pressure backwards over the lower front part of the arm. (3) Flexion of the forearm. If there be lateral as well as backward displacement, first apply lateral pressure to convert the diplacement into a simple backward type.

After reduction it is necessary to retain the arm in a flexed position for a couple of weeks. This may be neatly accomplished by a narrow figure of eight swathe to the elbow and trunk. The front of the chest, bend of elbow and axilla are powdered. Five or six feet are cut from a bolt of surgical gauze and folded into a strip about 3 in. wide. The operator stands in front of the patient. The forearm is flexed on the chest. The swathe is slipped behind the upper arm and forearm (Fig. 150) projecting beyond the latter a sufficient distance to be carried directly upwards over the shoulder of the same side and down the back to a point below the angle of the scapula (Fig. 151). The longer outer end of the swathe is then turned about the posterior surface of the arm, across the arm and forearm towards the unaffected side,

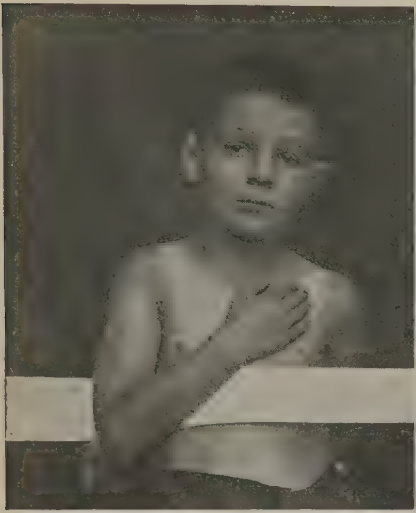

Fig. 150.—Folded gauze strip laid beneath flexed elbow.

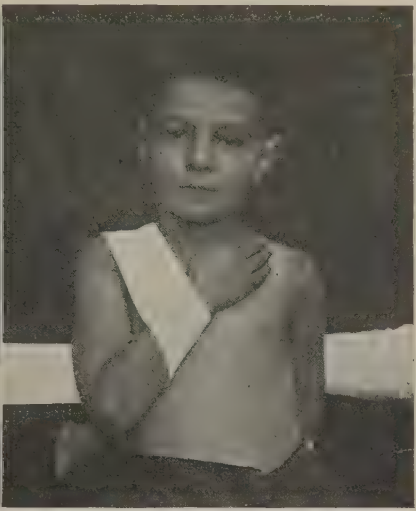

Fig. 151.—Mesial end carried over shoulder of affected side and down the back.

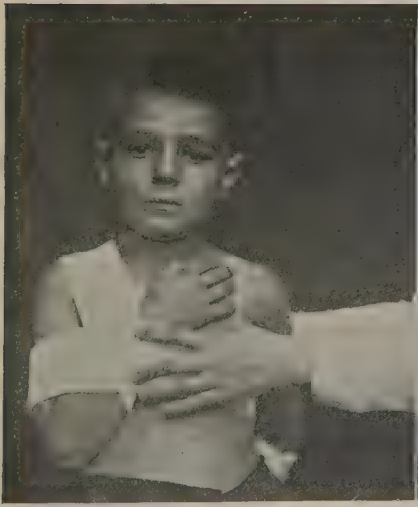

Fig. 152.—Other end of strip carried forwards across flexed elbow and completely about trunk until it overlaps in front.

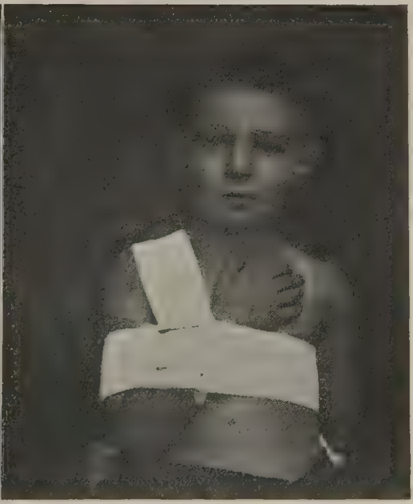

Fig. 153.—Strip secured with pins front and back.

then completely about the trunk until it overlaps itself just above the bend of the elbow (Figs. 152, 153). Here it is fastened with safety pins and the strip which was originally carried over the shoulder is fastened to it behind. The elbow should be soaked several times a day in hot water followed by gentle active motions within the limits of pain. Extension must not be carried beyond a right angle. During this time the arm may be carried in a sling. At the end of this time the sling may be abandoned, letting the arm fall at the side. Hot fomentations and active motions should be continued. Never should forcible passive motions be practiced. Although the joint may be a little stiff at first, as the normal repair processes progress, active motion will become less and less painful and the child will gradually regain full use of the arm.

Injury to the coronoid process demands no special treatment. Acute flexion after reduction is adequate treatment for separation of the internal epicondyle or tearing of the origin of the flexor muscles of the wrist and hand. If the olecranon epiphysis be separated, the arm after reduction should be held just within a right angle rather than in acute flexion, and adhesive straps may be helpful in drawing down the triceps and attached epiphysis. If the external epicondyle be separated care must be taken that the biceps tendon is not caught behind the humerus during reduction.

Dislocations of Both Bones of the Forearm.—Rare Types.—Although all of the following forms of dislocations of both bones of the forearm at the elbow have occurred in children, they are so rare that they will be but briefly considered. In all cases the diagnosis is to be made by identifying the two epicondyles, olecranon and the head of the radius and by noting their relations to one another. Inasmuch as considerable force is necessary for the production of these types, it is always important, if readily possible, to have a roentgen examination in order to detect a complicating fracture or epiphyseal separation. It has been noted under backward dislocations of both bones that there may be an associated lateral displacement. These are to be regarded as backward dislocations because the coronoid process and head of the radius lie either below or behind the humerus. Only those cases in which the coronoid lies in front of and the olecranon in back of the humerus are regarded as pure lateral displacements.

Inward Dislocations.—Reduction should be accomplished by outward lateral flexion combined with downward traction and direct pressure outwards over the ulna.

Outward Dislocations.—These may be incomplete or complete. Three types of the complete form have been observed. In the first the sigmoid cavity of the ulna embraces the lower border of the external epicondyle; in the second it embraces the outer border of the epicondyle; and in the third it embraces the supinator ridge of the humerus. In the last two the forearm is necessarily pronated. Curiously no mention

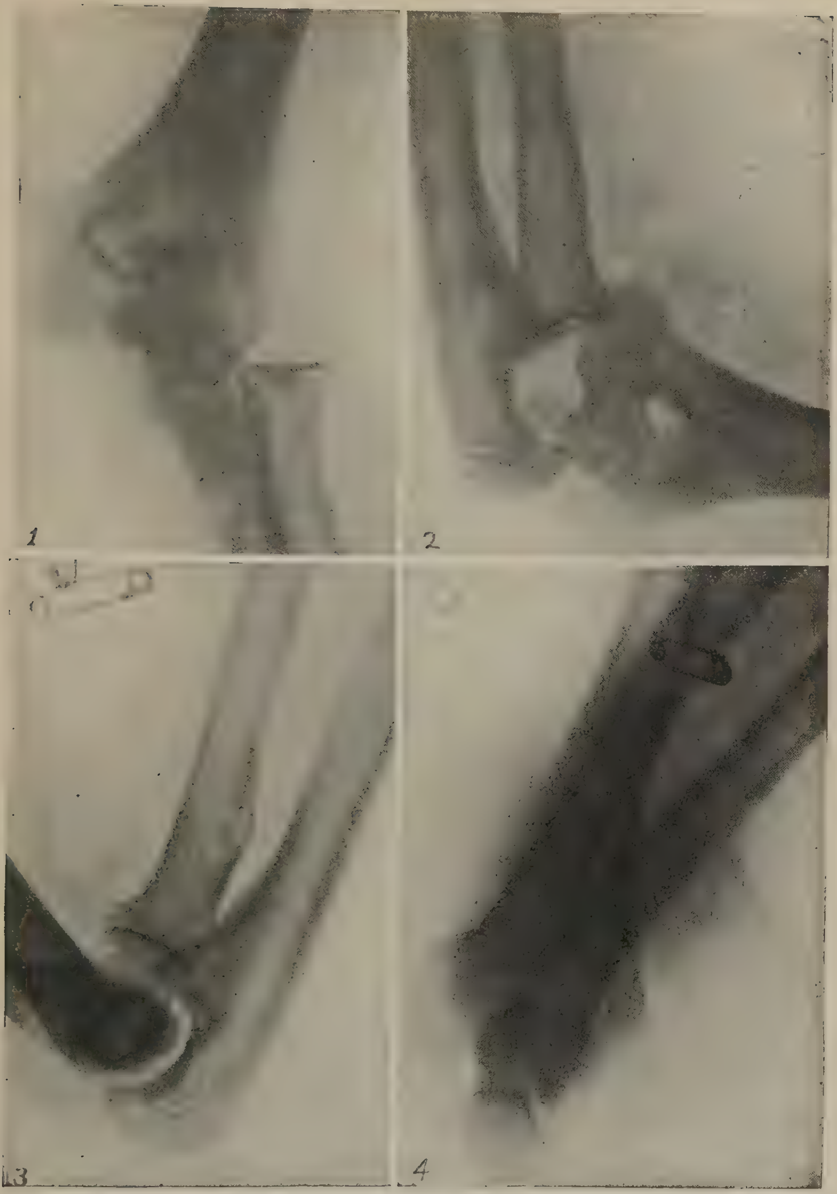

FIG. 154.—1, Lateral dislocation of elbow, anteroposterior view. 2, Lateral dislocation of elbow, lateral view. Note internal epicondyle torn off and lodged between joint surfaces. 3, Lateral dislocation of elbow after reduction. 4, Lateral dislocation of elbow after reduction. Note replaced internal epicondyle.

is made of separation of the internal epicondyle in the complete type whereas it has been observed a number of times in the incomplete type.

Inasmuch as cases of lateral dislocation of the elbow are rare, I herewith note a case seen Sept. 27, 1920, at the Massachusetts General Hospital.

A lad of 14 emerged from a scrimmage with several other boys. The left arm was injured at the elbow. I saw him a few hours later. The arm in the region of the elbow was moderately swollen. There was striking increase in the transverse diameter of the arm at this point. The head of the radius could be felt projecting beyond the external condyle. The olecranon was posterior to the humerus but moved outwards so that the sigmoid cavity embraced the capitellar surface. Flexion was limited to about a right angle and extension to about 45° beyond a right angle. An x-ray showed that the internal epicondyle had been avulsed and lay between the articular surfaces of the humerus and ulna. Under ether efforts to reduce by extension, inward pressure over the head of the radius, and flexion were futile. The following day through an anterior incision at the bend of the elbow, I readily removed the avulsed internal epicondyle from between the joint surfaces. Reduction was then readily accomplished, and as the exposure was sufficiently wide, the avulsed epicondyle was sutured in position. The arm was put up in acute flexion, motions within a right angle were promptly started. The boy made an uneventful recovery and has perfect function of the joint.

Reduction in the incomplete type should be accomplished by extension followed by direct inward pressure over the head of the radius. If the internal epicondyle has been separated and drawn into the joint, open reduction may be necessary. Reduction of the complete types should be accomplished by downward traction, supination and direct inward pressure on the head of the radius and olecranon.

Forward Dislocations.—In dislocations of the first degree the olecranon rests on the lower surface of the trochlea. The olecranon epiphysis is usually separated. In dislocations of the second degree the ulna is displaced upwards in front of the humerus. In some of these cases the ulna is fractured; in others the olecranon epiphysis is separated. The ulnar nerve is subject to injury. In the first degree type reduction is accomplished by downward traction on the upper part of the forearm followed by flexion. In some cases flexion alone suffices. In the second degree type downward traction is applied to the forearm flexed at right angles followed by pushing the forearm backwards with counter traction forwards on the lower end of the humerus. When fracture of the ulna exists it will usually take care of itself during the reduction of the anteriorly displaced radial head (Kirmisson[17]). In old cases in which the fracture of the ulna has united but in which the radius has not been reduced, replacement may be attempted by arthrotomy. Should this fail, the radial head should be excised (Ashhurst [18]).

Divergent Dislocations.—In the anteroposterior type the radius is displaced forwards and the ulna backwards. In the transverse type the ulna is displaced inwards and the radius outwards. In the former variety downward traction with the long axis of the forearm should be made until the ulna is reduced and then the radius pushed downwards into position with or without adduction and pronation of the forearm. The torn annular ligament may demand open reduction of the radius in recent cases or excision of the head of the radius in old cases.

Dislocations of the Ulna Alone at the Elbow.—The ulna has been displaced either backwards, inwards, or forwards while the radius has remained in position. The backward variety is very unusual but has been observed in children. The inward and forward types are extremely rare and perhaps have never been observed in children.

Backward Dislocation.—Three degrees of displacement have been observed: First, in which the coranoid process rests on the lower part of the trochlea; second, in which it has passed up in back of the humerus; and third, in which the ulna is displaced outwards as well as backwards behind the radius. Reduction of the first two may be accomplished by supination, abduction and hyper-extension. The third has been reduced by direct pressure but may require arthrotomy.

Dislocations of the Radius Alone at the Elbow.—The radius may be displaced forwards and rarely outwards or backwards unaccompanied by dislocation or fracture of the ulna. There is another subluxation of the radius which is much more common and confined entirely to children, the so-called dislocation by elongation (vide infra). The diagnosis of the forward, backward and outward dislocations depends upon identifying the head of the radius and its relation to the epicondyles and olecranon. In any there may be separation or injury to the upper radial epiphysis and in unreduced cases there may be subsequent disproportion in the lengths of the radius and ulna. Reduction of the outward, backward and forward varieties may be accomplished with the arm in full extension and supination either by traction on the wrist or adduction of the forearm combined with direct pressure on the head of the radius. If the capsule be interposed, arthrotomy may be necessary.

Dislocation by Elongation.—A child not over three or four years old is lifted by the wrist. He cries with pain, refuses to use the arm and holds it slightly flexed at the elbow and in pronation. Examination may reveal nothing but a little tenderness over the head of the radius and limitation in supination. Perhaps it may be observed that the interval between the head of the radius and capitellum of the humerus is greater on the affected side. This is the characteristic story of subluxation of the head of the radius in children. Probably the head of the radius is partially drawn down into the orbicular ligament. This is the theory of DuVerney,[19] and is the one generally accepted today.

Reduction is to be accomplished by supination with an upward thrust of the forearm against the upper arm followed by flexion at the elbow. No retentive apparatus is necessary but to satisfy the parents a sling may be worn for 24 hours. Doubtless this subluxation is sometimes unknowingly reduced by the child or parent on the way to the doctor's office or to the hospital clinic. In such cases except for the characteristic story and some tenderness over the head of the radius, nothing can be made out. Supination may be slightly resisted but can be completely accomplished.

DISLOCATIONS AT THE WRIST

It is with greatest rarity that violence at the wrist of a child is so directed as to produce dislocation. Traumatism in this region results commonly in separation of the lower radial epiphysis or greenstick or complete fracture of the radius or both bones of the forearm just above the wrist. This chapter is not concerned with the so-called congenital dislocations nor with the anterior luxation of the carpus of gradual onset (Madelung's deformity[20]).

DISLOCATIONS OF THE METACARPALS

Although one or several metacarpals may be simultaneously dislocated at their articulations with the carpus, backward dislocation of the first is by far the most common.

Backward dislocation of the first metacarpal may be caused by hyper-extension or hyper-flexion. The proximal end of the bone may be felt in the "anatomist's snuff box" resting on the trapezium which becomes abnormally prominent at the base of the thenar eminence. Reduction is readily accomplished by traction on the thumb and direct pressure. The thumb should be held in abduction and extension by plaster of paris or adhesvie strapping or an angular splint which fits either between the thumb and forefinger or between the dorsal aspect of the thumb and the radial aspect of the forearm.

DISLOCATIONS OF THE THUMB

Backward dislocations of the thumb at the metacarpo-phalangeal joint are especially interesting because they are so frequent and because they are so often difficult to reduce. Forward and outward types are usual. It is to be remembered that the joint is strengthened by an anterior and two lateral ligaments. Incorporated in the former are two sesamoid bones in connection with the attachments of the short flexor.

Backward dislocation is reduced by hyper-extension at the metacarpo-phalangeal joint. The anterior ligament may stretch sufficiently to permit the phalanx to slip upwards on the head of the metacarpal, or it may rupture permitting the phalanx to slip upwards past the head of the metacarpal onto its dorsal surface. As shown by Farabeuf,[21] the anterior ligament ruptures at its attachment to the metacarpal rather than at its attachment to the phalanx. Consequently when the phalanx passes upwards over the head of the metacarpal onto its dorsal surface, it carries with it the lateral ligament with the attached sesamoid bones. Either the sesamoids (according to Farabeuf) or the ruptured anterior ligament alone (according to Stimson[22]) may become interposed between the joint surfaces and prevent reduction. In the complete type the tendon of the flexor longus pollicis usually, but not invariably, is displaced to the inner side. In the incomplete type the proximal phalanx lies at an obtuse angle to the dorsum of the metacarpal. In the complete type it lies at right angles to the metacarpal. In both types the distal phalanx is somewhat flexed. The metacarpal itself is somewhat adducted and its head is felt prominently in the palm. During reduction flexion of the proximal phalanx before it has been brought down over the head of the metacarpal tends to engage the anterior ligament (or sesamoids) between the articular surfaces. Therefore relax the muscles of the thumb by carrying the metacarpal into the palm and push the proximal phalanx, still in its erect position, distally over the head of the metacarpal. This maneuver pushes the torn anterior ligament and sesamoids in front of the phalanx past the head of the metacarpal. If several efforts under anesthesia fail, it is better to resort to open reduction than to force. A longitudinal incision is made over the head of the metacarpal, the retracted and interposed anterior ligament identified, nicked and lifted from its position between the articular surfaces. A short posterior splint from the wrist to the tip of the thumb or molded gutter splint of felt or gutta percha may be used. The thumb may be flexed and held against the palm by adhesive strapping.

DISLOCATIONS OF THE FINGERS

The fingers may be dislocated backwards or forwards at the metacarpo-phalangeal joints, or backwards or laterally at the interphalangeal joints. In the backward dislocations the capsule may be torn away at its proximal attachment and, just as in the thumb, it may be interposed between the articular surfaces during reduction if simple flexion and traction is used. Moreover sesamoid bones may occur at the bases of the second and third fingers. Reduction should therefore be carried out as described under backward dislocations of the thumb. The displaced portion should be retained in its extended or hyperextended position and pushed or drawn well over the end of the proximal bone before flexion is attempted. After reduction the fingers may be held in position over a cylindrical roll in the palm.

Reduction of lateral displacement at the interphalangeal joints is usually readily accomplished by traction and appropriate pressure.

In no case of dislocation of the phalanges, especially one resulting from a blow, should a prognosis for complete anatomical and functional restoration be made until an *x*-ray has precluded a small chip of bone into the joint from an accompanying fracture.

DISLOCATIONS OF THE HIP

Traumatic dislocations of the hip unaccompanied by separation of the femoral epiphysis are unusual in children. The commoner dislocations, so-called congenital disease of the hip, are not within the province of this chapter. The commonest form of traumatic dislocation is backward and may be of the usual dorsal or the unusual everted dorsal type. Other forms of dislocation are: Downwards and inwards (obturator and perineal), upwards and forwards (pubic, pectineal, and suprapectineal), and the rare directly upwards (supracotyloid) and directly downwards (infracotyloid). Nathan Smith[23] was the first to formulate a rational manipulative procedure for reduction of the dorsal form. We are indebted to Bigelow,[24] however, for a scientific understanding of the types of dislocations and rational methods of reduction.

Backward Dislocations.—Dorsal Type.—As the name suggests, the head of the femur comes to lie behind and above the acetabulum. The capsule is torn in its lower posterior part and perhaps inferiorly. Both bands of the Y-ligament are intact. The leg is flexed at the knee and hip, rotated inwards and adducted. Abduction and external rotation are resisted. The summit of the great trochanter lies above a line drawn between the anterior superior spine of the ilium and the tuberosity of the ischium (Nélaton's line). Johnston[25] has reported a case which occurred in a six months infant seen for the first time six and one-half years later. The sciatic nerve may be somewhat pressed or stretched but is usually uninjured. Reduction is accomplished by the following steps: The anesthetized patient is placed upon his back on the floor; an assistant steadies the pelvis; the operator then (1) *flexes* the thigh to a right angle with the trunk preserving the adduction and inward rotation of the limb, (2) *lifts* the thigh to draw the head towards the acetabulum and the rent in the capsule, (3) *rotates* the thigh *outwards*. This renders the outer band of the Y-ligament and the posterior part of the capsule tense and swings the head of the femur forwards into the acetabulum. Traction should not be relaxed until the limb is rotated well outwards.

The gravity method advocated by Stimson[26] is more applicable to adults than to children. The patient is placed in prone position upon a table. The pelvis is drawn down so that it overhangs the edge of the table. The uninjured leg is supported horizontally by an assistant. The dislocated thigh is allowed to hang perpendicularly. The leg is bent at the knee and supported in a horizontal position at the ankle. The weight of the thigh and leg are thus used to overcome muscle spasm and the head of the femur gradually slips into position.

Everted Dorsal Type.—The symptoms are those of the dorsal type except that the leg is everted (rotated outwards). Bigelow has shown that the head escapes posteriorly and inferiorly with the leg flexed, adducted, and rotated inwards, but is at once rotated outwards tearing the outer band of the Y-ligament. The leg may be fully extended. Reduction is accomplished by flexing, rotating inwards, and adducting. This converts the dislocation into the common dorsal type and reduction is completed as such. Owing to the rupture of the outer band of the Y-ligament, the last maneuver, external rotation, is of little value.

The head of the bone will have to be guided into the socket by the operator while an assistant makes upward traction.

Dislocations Downwards and Inwards. Obturator or Thyroid.—As the name suggests the head of the bone comes to lie over the obturator foramen and reaches this position through a rent in the lower inner portion of the capsule. Powdrell[27] has reported such a dislocation in a six months infant. Woodward's[28] compound dislocation is probably of this type although the head and neck of the femur protruded through a wound 2½ in. below the angle of the pubes and just anterior to the adductor longus. This was a boy of 12 who had been rolled over and over in front of a moving freight car. He died of shock five hours later. The force is usually one of violent abduction or less frequently direct pressure from behind with the leg in abduction. The leg is somewhat flexed at the hip, abducted, and usually rotated outwards. Adduction and complete extension are resisted. The greater trochanter lies below Nélaton's line. The head of the bone may be recognized over the obturator foramen and if not separated from the neck, may be felt to turn upon slight rotatory movements of the thigh.

Reduction is accomplished by the following steps. The anesthetized patient is placed on his back on the floor. An assistant steadies the pelvis. The operator then (1) *flexes* the thigh to a right angle with the trunk, preserving the abduction and outward rotation of the limb; (2) *lifts* in the direction of the long axis of the thigh to draw the head towards the acetabulum and the rent in the capsule; (3) *rotates* the thigh outwards while still maintaining traction. (For the last step may be substituted inward rotation and extension.)

Perineal Dislocations.—As the name suggests the head of the bone comes to lie in the region of the perineum and may press upon the urethra or rectum. Macouchy[29] reports a boy of 14 who landed in a sitting position on the deck of a vessel after a fall of 60 ft. from a mast. The head of the femur protruded through the perineum. The boy died two days later from a coincident fracture of the skull. Inasmuch as the head of the femur is carried further downwards and inwards than in the obturator type, the flexion, abduction and external rotation of the limb are more marked. Adduction and extenson are resisted. The outer aspect of the thigh and buttock are flattened. The head of the femur may be felt in the perineum. Reduction is accomplished as in the obturator type. A greater amount of traction is necessary to bring the head to the edge of the acetabulum.

Dislocations Upwards and Inwards.—As the terms pubic, pectineal and suprapectineal indicate, the head of the femur comes to lie in these types either in the region of the superior pubic ramus, the ilio-pectineal eminence, or within the pelvis under or through Poupart's ligament. Although rare, all types have been observed in children. Perkins[30] has reported Langmaid's case; compound dislocation in a girl of eight with the head of the femur protruding through a wound near the middle of Poupart's ligament. She recovered with a stiff joint. The mechanism of these types of dislocations may be as follows: With the leg fixed, the body may be bent forcibly backwards (hyperextension of the thigh) or the limb may be first flexed, abducted, and rotated outwards, and then extended. In cases produced by hyperextension the rent is in the upper anterior portion of the capsule. In those cases produced by abduction and rotation, the head leaves the socket through a rent lower in the anterior part of the capsule and is subsequently carried upwards by extension of the thigh. In the pectineal type the limb is markedly everted and but slightly abducted. In the pubic type the limb is everted but also considerably abducted and flexed. In both types the outer and posterior portions of the hip are flattened and the trochanter is below Nélaton's line. There may be disturbance of circulation in the limb because of compression of the femoral vessels over the neck of the femur. Sensation may be disturbed by stretching of the anterior crural nerve. In the pectineal variety the thigh may be flexed and rotated inwards or extended and rotated outwards. Reduction is to be accomplished by *traction* on the thigh in the direction in which it lies; *flexion* of the thigh, perhaps with some abduction to permit the head to slip downwards; and finally inward *rotation* and *extension.* As the thigh is slowly flexed and perhaps abducted, it may be necessary for an assistant to seize the head of the bone and push it downwards.

Dislocations directly upwards (supracotyloid) have been very rarely observed in children. Persistence of the head of the bone below the socket (infracotyloid) is also extremely rare. The head usually passes forwards or backwards resulting in either an obturator or dorsal dislocation.

DISLOCATIONS OF THE KNEE

Dislocations of the knee in children are rare. Separations of the lower femoral or upper tibial epiphyses although producing displacement of the bones at the joint are to be regarded as fractures rather than as dislocations. The dislocation is possible only because the epiphysis is separated. On the other hand in the mechanism of a true displacement the epiphysis may be crushed by the moving bone or even vertically fractured, and the fractured piece driven in the direction of the displacement. In such cases the displacement is the primary lesion and the crush or partial separation of the epiphysis secondary.

Dislocations at the knee may be forwards, backwards, inwards, outwards, or by rotation. They are not infrequently compound, and, especially in the forward and backward varieties, the popliteal vessels and nerves are subject to injury. It is important therefore before and after reduction to test the pulsation in the dorsalis pedis and posterior tibial arteries. The vessels may be completely torn or the inner coat so damaged that thrombosis results. Cases in which the integrity of the vessels is in doubt should be kept under careful observation. Gangrene may ensue, requiring amputation. Disturbance of sensation may be noted soon after the injury or may not be observed until after several weeks when trophic ulcerations may appear. In compound cases the skin wound is almost invariably a transverse one in the popliteal space. Usually in the absence of injury to the vessels and nerves, debridement with immediate closure may be practiced.

Posterior Dislocations.—These dislocations may be produced by direct violence backwards on the front of the leg below the knee or forwards on the back of the thigh in the popliteal space, by hyper-extension of the leg, or by torsion of the leg (*cf.* having it caught in the spokes of a revolving wheel). It is interesting that these torsion accidents also are common causes for separation of the lower femoral epiphysis. The leg may be extended or occasionally flexed. The condyles of the femur are unduly prominent and the popliteal vessels may be distinctly palpable.

Reduction is to be accomplished by traction on the leg combined with pressure of the femur backwards and of the tibia forwards. The leg may be retained for several weeks on a ham splint or it may be temporarily placed on a Cabot posterior wire splint with side splints followed by a light plaster from just below the groin to just above the ankle.

In cases in which the lower femoral epiphysis is fractured transversely and the posterior portion carried backwards, replacement may be accomplished by bending the leg first outwards and then inwards over a wedge placed opposite the epiphyseal line. The posterior fragment is then manipulated into position while the knee is gently flexed and hyper-extended. The limb is then held in a plaster spica with the knee in hyper-extension. The tense popliteus and fascia are utilized to maintain the fragment in position.

Forward Dislocations.—These dislocations may be produced by hyper-extension of the leg, by direct violence backwards immediately above the knee or forwards in the lower part of the popliteal space, or by rotation (*cf.* catching the leg in the spokes of a revolving wheel, machine belt, etc.). In profile the leg is seen to project forwards, and in the absence of much swelling, the soft parts in the region of the knee are drawn into transverse folds. The femoral condyles and condylar notch are prominent posteriorly. The popliteal vessels may be distinctly palpable.

Reduction is accomplished by traction on the leg followed by flexion at the knee. Care should be taken not to stretch unduly the popliteal vessels.

Lateral and Rotatory Dislocations.—These are unusual varieties. Their recognition and reduction are so simple as to require no special comment.

DISLOCATIONS OF THE SEMILUNAR CARTILAGES

Dislocations of the semilunar cartilages are unusual in childhood. Any severe injury of the internal lateral ligament may result in displacement of the internal semilunar cartilage. Displacement of the external cartilage is rare. The characteristic story is a twist or wrench of the knee accompanied by more or less pain followed by inability fully to extend the leg, more or less synovitis, and localized tenderness over the inner cartilage.

Prognosis.—The prognosis depends upon complete reduction and immobilization in extension for a sufficiently long period to insure healing of the lateral ligament and the moorings of the cartilage. Recurrent attacks of dislocation with more or less synovitis are not uncommon. Sir Robert Jones[31] firmly believes that many of these recurrences can be prevented and permanent cures obtained if complete reduction and appropriate immobilization are instituted at the first injury. Too often the surgeon is content with *almost* complete extension. He then forcibly holds the leg in extension against a ham splint by firm bandaging. Not only is the reduction incomplete but the cartilage is compressed between the bone ends.

Treatment.—The maneuvers for reduction are as follows: Acute flexion, lateral deviation, rotation inwards and full extension. Acute flexion is painful but permits free internal rotation; lateral deviation separates the bone-ends; full extension holds the cartilage in re-position and favors healing of the lateral ligament. Not until reduction is accomplished will the patient be able to hold the leg *voluntarily* in *complete* extension. It may be well also to walk with the foot inverted and the toes in-turned to take the strain off the weakened internal ligament. Arthrotomy with excision of the damaged cartilage should be reserved for recurrent cases with acute joint symptoms.

Traumatic dislocations of the fibula, tarsus, metatarsus, phalanges, and of the ankle are rare in childhood. Congenital and pathological dislocations in some of these situations do occur but do not fall within the scope of this article.

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CHAPTER CXIII

CONGENITAL DISLOCATION OF THE HIP

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By congenital dislocation of the hip we mean a dislocation discovered during infancy or early childhood when there has been no injury remembered that could have been sufficient to cause a dislocation.

In some of these cases it is assumed that the head of the femur has never been in its socket. In other cases it is assumed that it has been in the socket, but has slipped out some time during interuterine life. In other cases doubtless it slipped out at birth. In other cases it is known to have been in place at birth and has slipped out at or about the time the child began to walk; and in other cases at some later time without any injury sufficient to dislocate a normal hip and without the patient being conscious of the fact.

The assumption that some of these femoral heads have never been in their socket is based solely on a few dissections made by a few men who have not had sufficient clinical experience with congenitally dislocated hips to know much, if any thing, about the subject.

The assumption that these hips become dislocated at some time during interuterine life is based on another assumption that the hips have been in place during part of the uterine life plus the theory of mechanical dislocation from position. The theory advanced some years ago by Oscar Allis of Philadelphia is the most plausible of these theories, but it is based on the unfounded assumption that in all of these cases the dislocation is backwards. Lack of clinical experience led to the assumption that almost all congenital dislocations of the hips were backward and upwards; whereas the truth is that the vast majority are dislocated directly upwards.

That some cases are dislocated at birth seems probable, particularly when the delivery of the child has been difficult and the feet came first and were pulled by the obstetrician. On the other hand it must be remembered that these cases show the same anatomical defects as do the cases where the birth was easy, the head presenting and the feet not pulled. Few obstetricians ever examine the new-born baby to see if the hips are dislocated; nor would they know it if they were dislocated. Two of the most eminent and experienced obstetricians in Chicago each delivered a child by pulling at the feet after having done a version, and did not discover the dislocated hips which were found by the orthopedist later on. The most eminent physician that I have ever known overlooked a bilateral dislocation for four years, although he knew that something was wrong with the child. He assumed that it was rickets. This is not surprising when we know that there are no symptoms in the newborn child to attract attention to a congenital dislocation of the hip, as there is in the so-called congenital dislocation at the shoulder. There is no obvious malposition; no restriction of motion; and no obvious shortening—nothing in fact by which one can discover the dislocation except by educated fingers. An *x*-ray picture may show the head apparently in place when it is dislocated forwards, but not yet pushed up by use of the limb.

Congenital dislocation of the hip is not usually discovered until the child begins to walk; unless congenital dislocation in a previous child, or some relative, or some other malformations leads to a very careful investigation; even then one may be in doubt or fall into error. I have seen two babies where soon after birth the hips were found to be dislocated. The femoral heads could be felt out of their sockets, and *x*-ray pictures showed them to be out of their sockets—quite different from the hip on the other side. In one case the prior child had been operated upon for congenital dislocation while the mother was "carrying" the child in question. In the other case a careful examination of the newborn child was made because a cousin had had a congenital dislocation. In both cases when the child was brought for operation at the proper age—two and a half years, the hips were no longer dislocated.

The very most that can be said, then, is that by congenital dislocation of the hips we mean those cases where the hips are found to be dislocated at some time in early childhood without any known or suspected injury as a cause of it.

All we know of the cause of congenital dislocation of the hips is that they are anatomically defective. That they are anatomically defective we know. We also know that the use of such a defective hip in walking with the limb in the position of adduction may result in dislocation; and it seems fair to assume that upward pressure of the femur, held in the position of adduction during interuterine life, may be a causative factor just as downward pressure of the weight of the body in standing or walking after birth may cause it, as we know it may. There are then three causative factors: An anatomically defective joint; the femur in the position of adduction; and pressure, either of the femur upwards or the pelvis downwards, or both. But it must be remembered that there may be serious anatomical defects in the hip joint and yet no dislocation ever occur. I have seen one case, a large man 56 years old who never had had any symptom of any disability at the hip joints, where the *x*-ray picture showed that in one hip one-fourth and in the other one-third of the articular surface of the femur—the upper portion—was not embraced in the acetabulum (Fig. 155).

Our knowledge of the anatomical defects is derived from a few dissections by men having little clinical knowledge of congenital dislocation of the hips, from quite a number of open operations for the replacement of dislocated hips, and from *x*-ray pictures. About all one learns at an open operation is what one feels with the tip of the examining finger, so deep in the wound lies the joint. One may feel a constricted capsule—hour-glass contraction—between the head and the socket, and the lower portion of the capsule drawn across the lower portion of the socket like a curtain—hymen—forming a kind of pocket. When men of great experience, like Bradford of Boston, operating by the open method, fail to find the real acetabulum and bore out a new one believing that they are enlarging the anatomical socket and discover the error only at postmortem examination, it is evident that little of anatomical value is learned when hips are replaced by the open method. *X*-ray pictures in young children do not show the shape of the femoral head and the socket because of lack of ossification. The ossified portion of the head may not appear to be more than a third, or at most half the size of the undislocated head; and the socket appears shallow. It seems to me that there is no reason to believe that deficient circulation in the head is the cause of this lack of ossification, as recently claimed by Adams of Boston, for the lack of ossification is as great in the socket as in the head of the femur, and there is no reason for assuming that there is defective circulation in and about the acetabulum. It is the lack of use in weight-bearing with bone in contact with bone that causes the delayed ossification. But whether the cotyloid ring is constricted too much to admit the head, or is enlarged, it is impossible to tell. In older cases the femoral head is

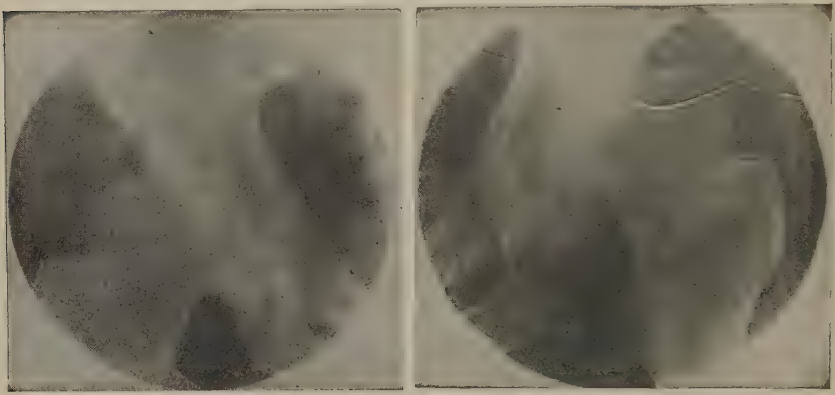

FIG. 155.—This man was a very large man; a policeman, 56 years old, who had never had any trouble with his hips. The hip abnormality was discovered accidentally when *x*-ray pictures were made of spine, pelvis and hips because of complaint of back pain. Note that at one hip one-quarter, and at the other hip one-third of the femoral head is not embraced by the acetabulum.

seen to lack the proper globular shape, and the bony socket is always shallow and enlarged, even when the femoral head has been replaced and has remained in place for so long as 10 years (Fig. 156).

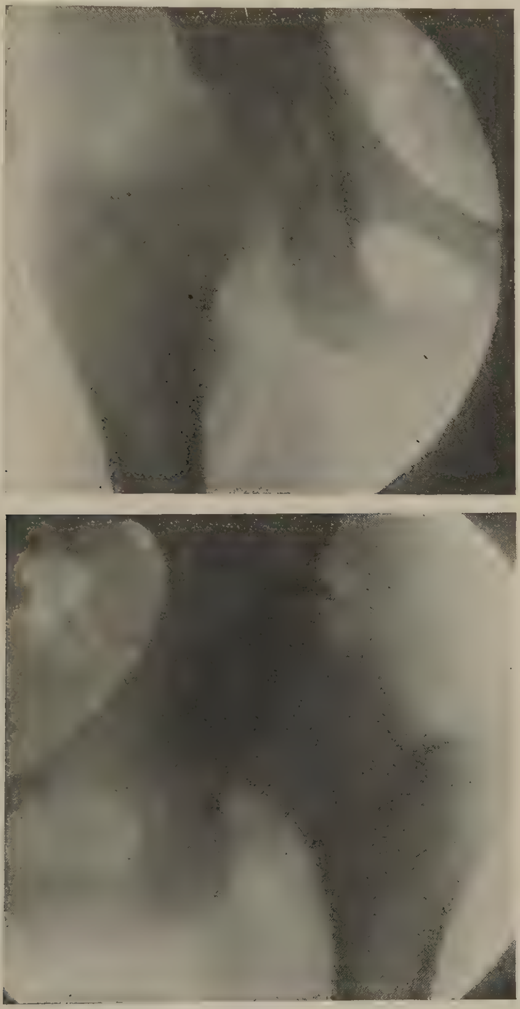

Fig. 156.—This young woman had her hip replaced by me when she was eleven years old. The radiogram was made nine years later showing the hip in place, but with a somewhat enlarged acetabulum and somewhat malformed caput femoris; but on clinical examination before the radiogram was made I was unable to determine which hip had been replaced. The appearance and function was the same as the other hip.

From all sources of information at our command it has been assumed that the femoral head is not normally globular. Some assume that the neck is twisted; but of this, more will be said later. The socket is shallow, and it has been assumed that because it has not been filled by the globular head, and because three bones form it, that it has developed into a more or less triangular depression. The capsule is elongated by as much as the head is displaced upwards, and more or less narrowed—"hour-glassed"—in that part of it which is between the head and the socket, and it forms a more or less tightly drawn curtain—hymen—across the lower portion of the socket (perhaps because of the pressure of the ilio-psoas tendon), obstructing the replacement of the head. It is assumed that the cotyloid cartilage is contracted—drawn in—since it has never embraced the head, so that it will not admit the head to enter the socket. The ligamentum teres is elongated by as much as the head is displaced upwards. Some open operators report that they have found it hypertrophied as well as elongated, and others that it is more slender than normal. There is some reason to believe that in some cases it is absent at the time of the bloodless operation, because femoral heads, broken off in an attempted bloodless operation, have gradually faded in repeated *x*-ray pictures as if absorbed from lack of the blood supply which the ligament carries.

There are no subjective symptoms in childhood. The objective symptoms in unilateral cases are only a limp and a short leg. The shortening when first discovered at 15 or 18 months may be only ½ in. At two and one half years it usually is about 1 in. At five years it is usually 1¼ to 1½ in., but may be 2 in. or even more. At 15 years of age it may amount to as much as 5 in. But the extent of the shortening depends in great measure on whether the head is dislocated forwards and upwards, or backwards and upwards, or directly upwards. Those displaced backwards and upwards go farthest upwards ultimately; and those dislocated forwards rarely go upwards more than 1 or 1½ in. Those dislocated directly upwards are between the two extremes. The limb may be smaller than the other as well as shorter. The range of motion at the hip joint in young children is normal and painless. In older cases full abduction may be wanting; and in forward dislocations inward rotation, and in posterior dislocations outward rotation may lack the full range.

In bilateral dislocations there is a "double limp" or waddle in walking. Because of the bony insecurity, the body is swayed from side to side as a duck walks. The buttocks are unduely prominent, and the lumbar spine bowed forward. The space between the thighs at the perineum is broadened. As the child grows older the thighs become more adducted, and flexed on the pelvis, and the legs flexed on the thighs.

On examination one tests the range of motion, and demonstrates the absence of sensitiveness and muscular spasm, and notes the upward displacement of the greater trochanter. In an undislocated hip in a child one can lay the index finger across the thigh with the anterior superior spine of the ilium above the finger and the greater trochanter below it. One feels for the femoral head: In anterior dislocations one can feel, and sometimes see, the femoral head in front in the neighborhood of the outer side of the anterior superior spine of the ilium, or a little lower. In posterior dislocations, following up from the greater trochanter, one finds the head beneath the buttock muscles. In upward dislocations the head can be felt in front when the thigh is rotated outwards, and at the back when the thigh is rotated inwards and flexed. The length of the limb should then be measured from the anterior superior spine of the ilium to the inner malleolus in unilateral cases, or by Nélaton's line in bilateral cases—drawing the tape from the anterior superior spine of the ilium across the outer side of the hip to the tuberosity of the ischium. In undislocated hips the tape should pass just at the tip of the greater trochanter. In dislocated hips the greater trochanter will be found to be above the tape, and the distance can be measured.

The condition most likely to be confounded with congenital dislocation at the hip is a congenital absence of the head and neck of the femur, which condition simulates dislocation in all its symptoms except that the femoral head cannot be felt by the examiner's fingers, and its absence is revealed in the *x*-ray picture.

An acute epiphysitis in infancy may leave a patient with much the same gait and posture as congenital dislocation; but one cannot feel the dislocated femoral head, and again the *x*-ray picture reveals its absence. Also one usually is able to find the scar of the evacuated abscess.

Dislocation at the hip due to infantile paralysis gives no history of limping prior to the attack of poliomyelitis, and also shows extensive paralysis in the limb.

A traumatic dislocation should give a history of the absence of limping before the accident, and an immediate disability following it. Nevertheless, in anatomically defective hips the accident producing the dislocation may be a very trivial one. Somewhat more than 30 years ago a boy 14 years old, living in Brooklyn, N. Y., while standing and bending backwards had "something happen" to his left hip. He fell to the ground; but was helped up, and after an hour walked home, about a mile. The surgeon called treated him for hip disease for about a year in bed with weight-and-pulley traction, and then sent him to me to have a hip splint put on. The boy walked with the leg rotated outwards and not at all flexed. The femoral head could be felt dislocated forwards and upwards. My opinion was not accepted by the surgeon on the case. An operation was done, and my findings confirmed.

I have not met with a case where a congenital dislocation was mistaken for a coxa vara. Such a mistake ought not to happen; for coxa vara usually occurs at about adolesence in fat children with undeveloped sexual organs, and there is a history of no limping during childhood.

Congenital dislocation has been mistaken for hip disease; but such a mistake ought not to be made, because the earliest symptom of hip disease is restriction of motion, to some extent, in all directions.

The early history of congenital dislocation of the hip is of no great value to us. The first record I have found of any knowledge of the subject in this country and in England was brought from France by Carnochan of New York, who published a book on the subject in 1852. The results of the treatment by the early French observers may be ignored. No claims of cures were credited until Dr. Buckminster Brown of Boston reported one case in 1885 treated by prolonged traction with the thighs partially flexed. Some years later Bradford examined the patient and found the hips still dislocated (personal communication). In 1889 I reported one case treated in bed with a Taylor long traction hip splint. But I know now that it was ignorance that led me to believe the hip replaced and that the "relapse" occurred simply because I never had the hip in the socket. In 1888 William Adams of London told me that he had five cured cases. But he did not show me the cases, and never reported them. Of course he was mistaken in thinking he had replaced the hips. The first real replacements were by Hoffa of Wurzburg, and Berlin, who did an open operation and deepened the socket. He was followed by Schede of Hamburg and Lorenz of Vienna and others, and in this country Bradford of Boston and Royal Whitman of New York led in doing the Hoffa operation. The results of the Hoffa operation were not satisfactory. Many replaced hips slipped out again; and most of those that remained in place were left without any useful range of motion.

Treatment.—The first permanent replacement without a cutting operation of which I know was Paci of Pisa about 1887. He pulled the limb downward and abducted it to the full normal limit. Some years later Lorenz popularized the "bloodless operation" and modified the maneuver. (His operation will be described later.) In this country Bradford in Boston, Whitman in New York, and I in Chicago led in this work in this country, doing as we believed the Lorenz operation. We had a few successful cases, and a large percentage of failures. In 1902, and again in 1903, Lorenz came to this country and demonstrated his method in many of the larger cities. The results of Lorenz's operations were not what they ought to have been. To some extent this was due to the fact that the conditions under which he operated were much less favorable than in his own clinic in Vienna. A few femora were broken, either at the neck or just below the greater trochanter, and several were left with temporary paralysis. In a considerable number of cases attempted he failed to replace the hips. Of the 29 cases that I followed to the final result in Chicago, only two had permanently replaced hips. This percentage of "cures" is far below what it ought to have been. The great good that resulted from the visits of Lorenz to this country was that not only the doctors but also the people learned through the newspaper furore over Lorenz that there was such a thing as congenital dislocation at the hip, and that some cases could be cured.

As to the operatons: I shall not describe the "bloody" operation of Hoffa nor shall I describe any of its modifications; because in my opinion, the operation is never necessary. Any hip at which the nerves and blood-vessels are sufficiently long to make safe a replacement by a cutting operation, can be replaced by a skilful man without a cutting operation. A cutting operation weakens the supports that are needed to retain the replaced femoral head, and increases the chances of it slipping out, provided, of course, the operation does not leave a stiff hip. When the result is a stiff hip, it is a secure hip. No one has brought forward any considerable series of results to disprove this statement.

Sherman's Operation.—The one advocate of the cutting operation in this country was Sherman of San Francisco. His operation, as planned, opens down on the femoral head in its displaced position. With the operator's finger inside the capsule the hour-glass constriction is slit, and as much more of the capsule, particularly the curtain or hymen across the lower part of the acetabulum, as may be necessary to place the cartilage of the head in contact with the cartilage of the socket. The socket is not enlarged, as Hoffa did. Apparently Sherman found that this is not necessary. The elongated and sometimes hypertrophied ligamentum teres is left untouched. The capsule is sutured (but Sherman ceased to suture his capsules—it is

Fig. 157.—This is the case in which I replaced both hips for the late Dr. H. M. Sherman of San Francisco. He cut down on the right hip to learn just what had happened at my bloodless replacement. The radiogram was made five years later showing a perfect result in both hips.

too difficult to be worth while), and the wound is closed. Two months later Sherman did the second stage of his operation. On the theory that the femoral neck is always twisted, and always twisted in the same direction, he did an osteotomy of the shaft of the femur, and thought that he turned the lower fragment outwards so that when union takes place and the foot is once more turned to the front the femoral neck with its head will be turned more directly backward into the acetabulum. Sherman has not proven his belief that the femoral head is always turned too far forward nor have I proven my belief, that if the femoral neck is bent at all it must turn the head too far forward when the hip is in anterior displacement; turn it too far backward when the hip is in backward displacement; and bend it upwards, if it is bent at all, when the head is displaced directly upwards. There are very few hips with head dislocated to the front of the socket; there are more with the head at the back; but the vast majority of cases are neither back nor front—they are superior displacements. Why, then, operate on all cases as if the head were bent to the front? Sherman reported one case, a bilateral dislocation, in which he believed that his operation was necessary (both stages of it) on both hips. He let me replace the right hip, and then cut down on it to see what had happened. Then he asked me to replace the other hip (both of my replacements were bloodless); and although he believed osteotomy and twisting necessary, at my request it was not done. At the end of five years he reported both hips perfectly in place (Fig. 157).

Lorenz's Operation.—The Lorenz operation as done by him in this country in 1902 and 1903 was as follows: A rubber pad was placed between the child's legs to protect the perineum (at one time when this was not done the perineum was torn through from the vagina into the rectum), and over the rubber pad was placed the middle of a sheet folded diagonally with the ends tied at each side of the head of the table. Then one or two, or three assistants seized the leg, and bracing their feet against the table, pulled in unison. Then the leg, straight at the knee, was flexed until the foot lay beside the child's ear to "stretch the capsule." The capsule was also stretched by carrying the leg backwards, and from side to side. Then the thigh was partly flexed and abducted while the operator hacked at the stretched adductor muscles with the edge of his hand until they gave away. Again the leg was stretched by pulling it downwards. Now the operator was ready to attempt the replacement: With the region of the buttock back of the femoral neck resting on the wedge block of Koenig for a fulcrum, and the opposite side of the pelvis held down on the table by one or two assistants, the operator siezed the flexed knee, flexed the thigh to a right angle, and abducted it. The head entered the acetabulum over its posterior edge—unless the bone broke. With the thigh, then, flexed to 90°, abducted 90°, and rotated outward until its outer surface looked backwards a heavy plaster cast was put on extending from the lower border of the inner condyle of the femur at the knee up on to the body to the free border of the ribs.

There have been many modifications of this maneuver: Hoffa used the edge of the table in place of Koenig's block. G. G. Davis of Philadelphia placed the child face down on the table and, with the flexed knee raised, pressed downward on the region posterior to the joint. Bartlett, a lawyer of Boston, devised and patented a machine making powerful traction while a lever pried up against the head and neck of the femur. Bradford devised a simpler (and unpatented) apparatus aimed to act in the same way. In all these methods it is obvious that no attempt is made to know in just what relation the femoral head lies to the acetabulum at any stage of the replacement. The operator pulls and pushes and pries over a fulcrum or with a lever, purely by guess as to the relation of the head which he is moving about to the socket into which he is trying to place it, like a blind man trying to thread a needle without even resting one hand against the other. For two years I used the Lorenz method, and during that time I broke the femoral neck in five cases. Then I developed a more intelligent method.

Ridlon's Operation.—In the author's method there is no preliminary stretching of the leg in any direction. Why, because in the first place it is unnecessary; and in the scond place stretching weakens the muscular support of the replaced head; and, third, stretching as Lorenz did sometimes causes paralysis. There is no hacking of the adductor muscles; because their support is needed to prevent the replaced head from passing inwards and forwards into the position of "anterior transposition;" and because the hacked and torn adductor muscles sometimes bleed subcutaneously to an alarming extent—even death may result from this cause. Koenig's wedge-block is not used as a fulcrum, prying over which it is not difficult to break the neck of the femur. The femoral head is not forced in over high posterior border of the acetabulum; but is coaxed in at a lower point, almost skidded in over the ischiatic portion of the socket.

The steps of my operation are: The hips are raised from the table and rested on a sand-bag or folded sheet; the assistant holds down the other side of the pelvis; operating on the left hip, the operator takes the flexed knee in his right hand, passes his left hand into the child's crotch with the fingers at the back and the thumb in front. The index finger finds the head, the middle finger the neck and the ring finger the greater trochanter; the thumb rests in front of the acetabulum, about 1 in. below the iliac spine. Then with the right hand grasping the knee the thigh is fully flexed. The fingers of the left hand keep in contact with the head, neck and greater trochanter and inform the operator of the exact position of the head as it goes downwards with the flexing of the thigh. In some young cases with less than an inch of shortening full flexion of the thigh may throw the head too low, even back of the ischium and into the obdurator foramen. In other and older cases with

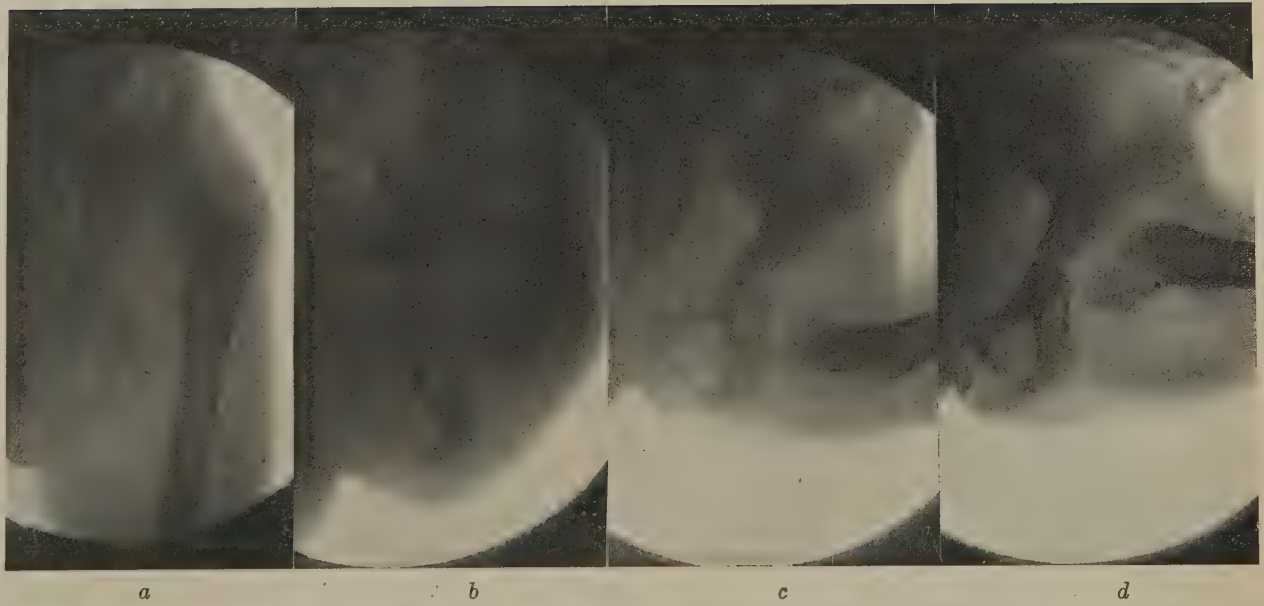

Fig. 158.—*a*, A relapse case, showing quite a deep false socket, with an upward displacement of about 1¾ in. *b*, With the thigh fully flexed—the first position during replacement—showing the head thrown well down and back of the lower part of the acetabulum. *c*, The second position; as the head rises up and passes into the socket. *d*, The head in the socket (and limb in the Lange position of right-angled abduction without rotation).

greater shortening—2 in. or more—full flexion of the thigh does not throw the head low enough and it is necessary to rotate the thigh inwards by turning the foot and leg (below the knee) outwards (Fig. 158). When the fingers on the head, neck and greater trochanter and the thumb in front of the socket inform the operator that the head is in the desired position, the thigh is abducted slowly with gradually increased force. Presently the head will be felt to come up under the thumb which rests in front of the socket. The abduction is slightly relaxed, and the head sinks back; again the thigh is abducted, and again the head comes for ward. In easy cases the head then pops in. In more difficult cases it is well to let the head sink back and then bring it forward several times, else some fibers of the adductor muscles be torn; or possibly the neck broken; which accident, however, has not happened to me when operating by this maneuver. In many cases the head does not enter the socket with an audible pop, but with a distinct jolt. In other cases there is not even a jolt; but the operator knows by the position of the head under his thumb that the head is in the right place, looking through the constricted capsule into the socket. If then he adduct the thigh the head jumps out the socket over some part of the upper portion of the rim. Again the head is replaced and bored into the socket. This may be done several times until the constriction of the capsule is stretched and the jump when the head goes out is more pronounced. In many cases if the hand is removed from the knee the thigh will adduct of itself and the head slip out; in other cases the hand can be removed from the knee and the head will not slip out. Such cases are sure to give perfect results. It is often well to try whether the replacement is more secure in the Lorenz position—outer side of the thigh looking backwards—or in the Lange (Munich) position with the back of the thigh looking backwards. In most cases it will be found to be more secure in the Lorenz position. Then the Lorenz plaster spica is put on. But if the Lange position is chosen the cast must be extended nearly to the ankle with the knee flexed to a right angle. It is believed that the Lorenz position tends to an anterior transposition of the head; and that in the Lange position the head looks more directly backwards into the acetabulum, and tends somewhat more to its slipping out backwards. The important thing, however, appears to me to be that with the leg in the Lorenz position the child can walk in from two to four weeks, while with the leg in the cast with the knee bent the child cannot walk at all. Walking on the replaced hip is absolutely essential to secure a perfect result; and whenever I put up a hip in the Lange position I cut the leg portion of the cast off to the Lorenz length at the end of two months so that the child may walk. When this is done the thigh rotates inward to about a mid-position between that of Lorenz and Lange. In neither of the three positions have I had the head slip out while the cast was being worn. This is probably because for the head to slip out backwards the thigh must come forwards; and to go upwards the thigh must be adducted, both of which movements are prevented by the cast. It may go out in front if the knee is carried too far back—back of the transverse plane of the body, or if the Y-ligament has been badly torn during the manipulations (Fig. 159).

The hip being replaced, a snugly fitting garment of stockinette (tubular webbing) is put on the leg and body, into which have been drawn two strips of muslin bandage, for "scratchers;" over this sufficient sheet-wadding bandages are wrapped to pad well all bony prominences. This is bound firmly and smoothly down by muslin bandages and then a thick plaster cast is applied. The most valuable thing that I learned from Lorenz was to put on a *thick* cast. It should be a quarter of an inch thick at the knee and half an inch thick about the hips and the body, so that one need not worry about its breaking during the eight months that it is to be worn. Slow-setting plaster bandages should be used so that the edges can be rubbed smoothly rounded after the cast has been trimmed. Finally, stockinette is drawn over the outside of the cast and sewed in place, and a soft towel is drawn between the child and the cast at the back and folded and pinned over a pad at the back of the body portion of the cast of sufficient thickness to raise the cast and the child up from the bed, so that the child lies *in* the cast, and not the cast *on* the child. If this is not done and the cast sinks into a soft bed it may press painfully against the

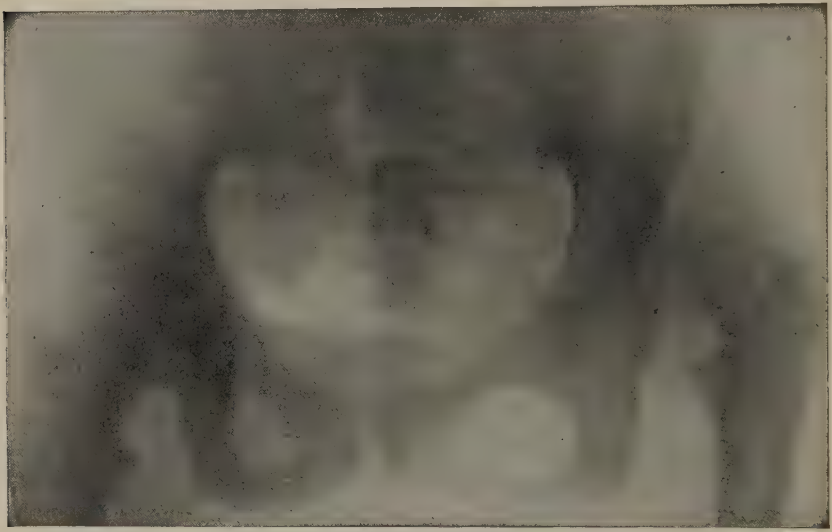

FIG. 159.—A "good result." This hip was replaced in 1896 and was in place when the cast was removed at the end of eight months. She was not seen again until eight years later. She stood straight and walked with only a perceptible limp, but the radiogram shows a typical "saucer" socket too shallow to retain the head of the femur.

inner condyle at the knee and even cause a pressure-sore. The towel and pad are used only while the child remains continuously in bed. In the nursing it is important to see to it that the cast is not soiled with urine and feces. The child should spend some time every day lying face down, and may be turned and moved, and taken up without fear of displacing the hip. As soon as the child can be taken up without crying from pain it can leave the hospital and go home. In the average case this is three or four days. Some easy cases go home the day of the operation, even to a distance of 200 miles; but difficult cases usually remain in the hospital about a week. After that I give the case no further attention, beyond urging the child to stand and walk as soon and as much as possible; and rarely ever do I see these children after they go home until it is time to remove the cast at the end of eight months. I do not have them use the elevated shoe recommended by Lorenz. In some bilateral cases they use a modified "kiddy-car" a—straddle of which the child sits. It should be of such a height that the child rests the feet on the floor and uses them to push about the room. Difficult bilateral cases often cannot be induced to walk before the end of the second month; but walk they must sooner or later if the result is to be satisfactory.

In the replacement of a congenitally dislocated hip it is my opinion that only rarely is the cartilage of the head actually in contact with the cartilage of the socket. Contact is prevented by the contracted and folded-in capsule and by the elongated, and perhaps hypertrophied, ligamentum teres. For these reasons, and perhaps at times from a more or less triangular socket, or drawn-in cotyloid ring, the head simply looks into the socket, and is not really in it, cartilage to cartilage. Hence, the importance of early and much standing and walking, and use of the joint, to stretch the contracted capsule and ring, reshape the deformed head and socket, and lead to taking up of the slack of the elongated capsule and ligamentum teres.

The desirable age for operation is from two and one half to five years; but the age is of less importance than the extent of the displacement. No child should be operated on until "house-broke," that is, until the diaper age has passed; for the cast must be kept dry and clean.

No child should be operated on before the shortening amounts to 1 in.; for until then the muscular shortening is not sufficient to retain the replaced head securely in place. No child past the age of five should be attempted by one with small experience if the shortening amounts to much more than 2 in. To be sure I have replaced one hip where the shortening was 3¼ in.; but the suffering of the child was great for the first 10 days; my worry was not a little; and the result was a hip with little useful motion. I have replaced unilateral dislocations in two cases at the age of 13 years in which the shortening was not above 1½ in. (and other surgeons have reported replacing older cases), but the hips in my cases were left quite stiff. Bilateral cases are less favorable than unilateral cases, for the sockets are usually more defective, and in bilateral cases one replaced and one relapsed hip, as frequently happens, leaves the child more disabled than two dislocated hips. I replaced two hips in a large girl 10 years old, one 2⅛ and the other 2¼ in. short. It was two years before the legs could be brought together. Nine years later the hips were found to be in place and she walked without any limp, but the range of motion was so little that she went up and down stairs with difficulty. As a general rule the more difficult the replacement, the more certain are the hips to remain in place, the longer will be the period of treatment after the cast is removed, and the more restricted will be the range of motion. Conversely, the easier the replacement, the more likely are these hips to slip out again.

When these hips do slip out during the early months after the cast has been removed, they ought not to be replaced at once, for they will surely slip out again. The operator should wait until the shortening has again reached 1¼ in. before replacing the hip again. Years ago in a bilateral case I was induced to operate too early. One hip stayed in, and the other slipped out. A year later I replaced the relapsed hip. It slipped out again. After waiting two years I replaced it the third time. Again it relapsed. Then I waited *five* years; replaced it; and it stayed in.

Occasionally a child grows so much during the period that the cast is being worn that the thigh is constricted and the leg swells. If this does not happen before about the end of the seventh month I direct that the cast be cut open and gapped from the crotch to the top of the knee; but if the swelling of the leg comes earlier, I put on a new cast.

When the cast has been removed, I direct the parents to rotate the leg inwards and adduct it, and also carry the flexed knee towards the shoulder of the opposite side. This is usually all that is necessary to restore the position and possible range of motion in the course of six months. I caution them that if the leg quickly returns to its normal position and range of motion the hip has probably slipped out. But in very difficult cases the hip refuses to come into adduction and must be stretched and adducted and again put into a cast for a couple of months until the structurally shortened muscles have lengthened.

The treatment of Denucé as described by Z. B. Adams of Boston, is as follows: "It is not very unlike the method of Dr. John Ridlon, but it is not the same, as the reduction is preceded by a manual stretching of the adductor muscles, and of this Dr. Ridlon does not approve.

This stretching is done by gentle stroking of the adductors with the soft part of the palm of the operator's hand—beginning at the pelvis and stroking downward in the longitudinal axis of the thigh, the skin having been covered with powder. I may say here that, in Professor Denucé's reduction and treatment, he lays great stress upon the point that no violence or force is to be used. As the adductors are stretched, the thigh is brought up to right-angle flexion and full abduction. Having stretched the adductors so that the thigh will lie upon the table at right angles with the trunk—the patient in the decubitus position—the attempt at reduction is then made. This is done by the operator standing on the side of the patient on which is the dislocated hip to be reduced; the thigh is flexed on the trunk—the knee being carried across in the direction of the axilla of the opposite side, and the thigh firmly pressed down on the anterior body wall. The knee is held in this position, a slight push being made in the longitudinal axis of the thigh. The fingers of the operator's other hand are placed under and around the head, neck, and trochanter, lifting up, and pressing the muscles in between the head and side of the pelvis; then circumduction is begun. The knee is carried across the body, trunk to its own side, and then downward to the surface of the table, and then abducted—all of this time the fingers behind the head are held firmly in place and lifting; the circumduction is slowly continued until the thigh is brought to a right angle with the body trunk and lying on the surface of the table—the assistant holding down the opposite side of the pelvis by a hand over the crest and anterior spine. The head is felt to *slowly* come forward and lodge in the acetabulum. As some of the doctors have suggested, it "oozes" in. At times, the shock is very slight.

After the reduction, the hip is tested for stability—the leg being drawn straight down; the point where the head jumps out is noted, and again the knee is brought up by being adducted at 90° flexion, and the point at which the hip jumps out of the socket is noted. Should the hip be very unstable, the plaster is applied with the thigh in a modified Wernsdorff position—that is, 90° of abduction and 110° of flexion; but the ordinary case, with fair stability, is put up in what Professor Denucé calls "ninety-ninety," that is, 90° of flexion and 90° of abduction. Over a single layer of bandage made of eiderdown cloth, and applied with the nap in, one layer of plaster bandage is put, and then a sheet of plaster of Paris, made from six thicknesses of crinoline, is moulded around the child, and this is fitted down and held in place by another layer of plaster bandage. The plaster extends from the nipple line to just above the ankle.

The hip being reduced and put in plaster, Professor Denucé sends the child home, often immediately after the operation, allowing simply time for the plaster to set. There is no trauma, no ecchymosis, no danger, and no pain afterwards—providing the child is young. He sees the case again at the end of three months. The old Sister gives the parents directions about the care of the child in the plaster—not to allow it to get the cast wet with urine, and to keep the child not in bed, but on the top of a flat, firmly padded table.

At the end of three months, if the child has grown rapidly, and on palpation the head of the femur is felt being pushed forward into the groin, the plaster is cut off above the knee, and the lower end of the femur and leg allowed to go free. The plaster is left intact, excepting for this change, and if the child has not grown too fast, the plaster is simply patched if soft, and allowed to remain in its entirety. At the end of six months—and Professor Denucé does not vary the time with variations in age—the plaster is bivalved into an anterior and a posterior valve, the child being bandaged into the posterior plaster, and the anterior plaster being thrown away. The parents are instructed by the old Sister in Professor Denucé's method of after-care.

Each day the child has an hour of hot sand, in a cloth bag, applied at the knee and over the front of the hip while lying in the plaster. After this heating, the child is coached to move its legs, to lift the femur forward, to straighten the knee, and to rock the lower leg. General heliotherapy is also given, beginning with periods of five minutes, and increasing the period daily five minutes until the child has one and one-half hours of sunning each day. At the end of two or three weeks, the posterior plaster is removed, and the child lies on the top of the firmly padded table.

The exercises are given twice a day, for a period of one-half hour each. No passive motion to the hip, but active motion; motion in abduction as well as adduction, and motion in rotation, flexion, and extension. The child is turned on its face, and taught to contract the gluteal muscles on both sides, and to rotate the thigh inward and outward while lying on its face, and to lift the thigh and hips off the table, and to swing the hips from side to side when lying prone on the table.

When the exercises have continued for three or four weeks, the child is given baths in a strong salt solution.

No weight-bearing is permitted on these hips until the center of ossification of the upper epiphysis of the femur is seen to have begun to increase in size markedly, and the head to shape itself, which is usually three to five months after the removal of the plaster. Then walking is begun, with very short intervals of standing, and gradually increasing the time the child is allowed on its feet. The exercises, local heat, and baths are continued for many months; cases of 31 months of exercise are recorded. The function is almost perfect in all of the cases." (Journal of Bone and Joint Surgery, 20:523, 1922).

As to **prognosis,** one may not quote percentages of results as *facts;* for to be of any real worth one must count cases in hundreds, all selected on the same basis and operated by the same surgeon in the same way. There has not been published any such group of cases. Every operator improves in skill as he operates on more and more cases; and with increased experience he rejects more and more cases as unsuited for operation. Eighteen years ago I attempted to reduce many hips that now I would not touch. In selected cases, namely, those not too young and those not too old, and those with enough shortening and those with not too great shortening, I think that my cases will show approximately the following percentages: Posterior dislocations from 80 to 100 per cent.; anterior dislocations not more than 50 per cent.; superior dislocations vary between the two as they more nearly approach the anterior or the posterior positon—these for unilateral cases in girls. Boys are more difficult, and the percentage of perfect results is not as great as in girls. In bilateral cases not

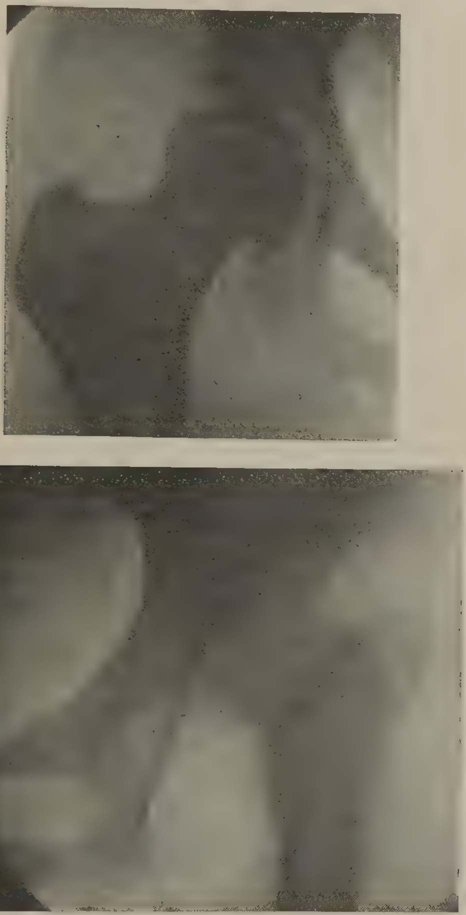

FIG. 160.—This is the case that Lorenz failed to replace and I replaced. On Oct. 24, 1914, twelve years from the day of the operation this young woman walked into my office without a limp. The joint motions appeared to be normal, but she would not permit an examination. Somewhat later I obtained these radiograms of the normal and the operated hips.

Note the further enlargement of the upper part of the socket, and the coxa-vara-like appearance of the femoral head and neck.

The young woman thinks that she has a perfect hip.

more than 50 per cent. give perfect results in both hips. By perfect results I mean cases in which the deformity and shortening are corrected, and 8 or 10 years after the operation it is not possible, without an *x*-ray picture, to tell which hip has been operated upon.

In such cases the *x*-ray pictures show the hip in its socket, but with the head somewhat deformed and the acetabulum shallow and enlarged. The cases that are not perfect ought to be what Lorenz called "good" results, namely, the lordosis, the prominent hip, and the flexion of the thigh corrected; the patient standing straight and walking well with little or no limp; but with the femoral head lying

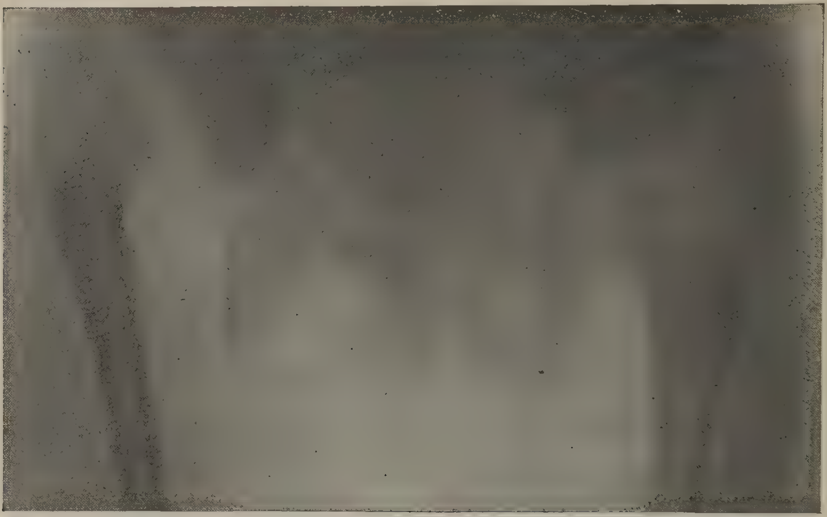

Fig. 161.—Before operation.

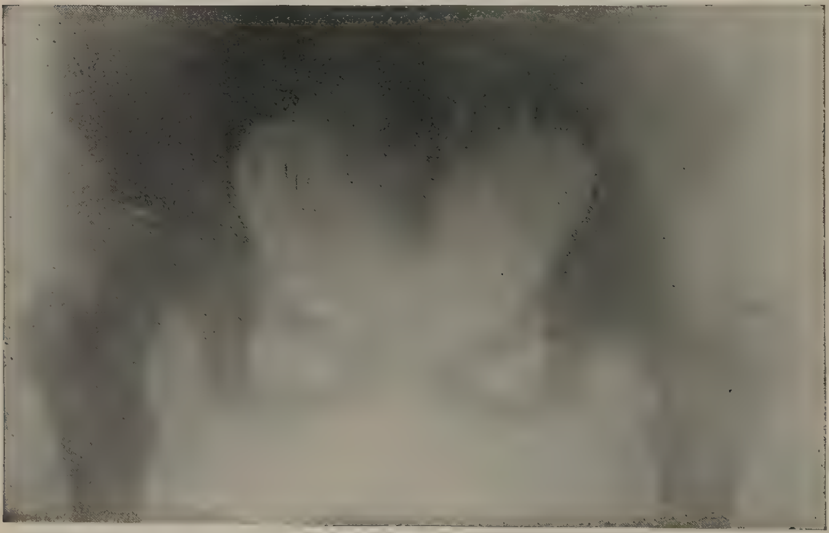

Fig. 162.—Result eight years after operation on case shown in Fig. 161.

in front and 1 to 1½ in. above the socket, with corresponding lack of length of the leg. Many of these young women believe that the hip is perfect. But if one operates on hips with too great shortening, as I have done many times, the perfectly replaced hips may be quite stiff; the femoral heads may show malformation from pressure, or may be depressed into the position of coxa vara (Fig. 160). This depression of the head may take place after the cast has been removed. One such was a case that Lorenz failed to replace, but which I replaced before she came out of the anesthetic. When the cast was removed at the end of eight months the hip was in place and the femur showed no deformity. Twelve years later, to the day, after the operation she came to show me her "perfect" hip. She walked without a limp and there was no restriction of motion; but the leg was short and the *x*-ray picture showed the head depressed.

In bilateral cases it should always be remembered that one hip is better than the other, but that both hips are and always will be defective hips, so that if one hip slips out and later on is replaced the good hip must be protected by a plaster cast in the straight position while the child walks with the re-operated leg abducted. Otherwise the hip that was in place, the previously replaced hip, will slip out when used in walking with the recently operated-on leg in abduction. Even hips that have never been dislocated may slip out when walked on in the adducted position after the other, the dislocated hip, has been replaced. I have seen three such cases—cases that just escaped being congenital bilateral dislocations. As there are cases with sockets so shallow that the replaced heads can never be made to stay in place, so there are cases where the sockets are barely good enough to retain the only partly included head when put to no abnormal strain. An anatomically perfect hip is one that will not suffer a dislocation without a fracture of some portion of the socket rim (Figs. 161, 162).

CHAPTER CXIV

WRY-NECK AND POSTURAL DEFORMITIES OF THE SPINE

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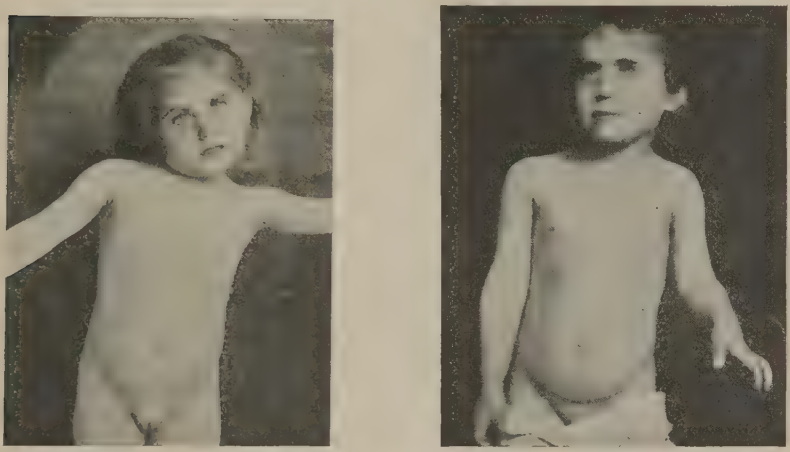
WRY-NECK

Wry-neck, spoken of commonly in orthopedic literature as torticollis or caput obstipum, implies a distortion of the cervical segment of the spinal column by reason of which the head is turned held to the one or other side of the body. Strictly speaking it should be regarded, not as a clinical entity, but rather as the result of an underlying abnormal condition either congenital or acquired. This concept is of importance inasmuch as its observance will tend to prevent no little diagnostic confusion. In the case of the congenital forms of torticollis the recognition of the underlying cause is of importance chiefly with respect to treatment, the deforming process having come to its conclusion for the most part; acquired torticollis, on the other hand is very likely to be the effect of a pathological process which demands careful recognition for the reason that it is still active, progressive and even destructive in character. The correction of the disfigurement which is involved may be, on this account, the lesser of the tasks which confront us, our primary duty being the control of a disease which may well otherwise menace the patient's life.

Congenital Torticollis.—As usually seen, congenital torticollis is a deformity produced by an abnormal shortness of the sternomastoid muscle of one side of the neck; in this both the clavicular and the sternal portions of the muscle are involved even though not always in equal degree. As a rule this muscle is the only one which is structurally shortened unless the patient has reached the later years of childhood; in this case other lateral muscles may be short as well as the other soft tissues of the neck.

From an etiological point of view, we know but little that is definite about this deformity. It has been attributed to intrapartum trauma; occasionally, we see hematoma of the sternomastoid muscle and especially after instrumental deliveries, but these cases do not go on to the development of wry-neck. The assumption by Mikulicz and Kader of the previous existence of an interstitial myositis with a subsequent fibrosis, is not borne out by the examination of the muscle during operation by various surgeons, the writer included. The explanation which seems most plausible for the usual form of this deformity is a position of the head in utero from which it does not move, so that the muscle of the one side does not grow as long as that of the other. This is an explanation which may serve as well for certain other congenital deformities, such as club foot, with which it is not seldom found associated. Very rarely the abnormal position of the head must be ascribed to malformations of the cervical bones, such as synostoses between the vertebræ or supernumerary vertebræ; cervical ribs have also been looked upon as a cause. However, these are not the usual cases of torticollis and they are to be recognized by the clinical examination, made with ordinary care.

Symptoms.—The characteristic symptom is the peculiar position of the head, which in the usual muscular form is to be explained by the abnormal shortness of the sternomastoid muscle. The head is thus *turned* toward the normal side and *inclined* toward the side of the short muscle. The chin points away from the affected side. The short sternomastoid stands out as a prominent ridge on the neck in contrast to that of the other side. Movement tending to exaggerate the deformity is easily made, whereas movement in the sense of correcting it is resisted by the too short muscle. According as the structural shortening involves the sternal portion, to a greater degree than the clavicular, the turning of the head as shown by the position of the chin will be greater than the inflection of the head as shown by the approach of the ear toward the shoulder, and *vice versa.* In case the deformity is caused by bony malformation, the mobility toward the normal side is likely to be interfered with and the cause will be demonstrated by the *x*-ray examination.



Figs. 163, 164.—Congenital wry-neck before and six months after operation.

In proportion to the age of the child secondary changes of the skeleton will be apparent. These involve all of the bones of the skull to some extent, being most evident in those of the face so that a definite asymmetry is to be seen in all older children. The side of the face toward the shortened muscle appears distinctly shorter than that of the opposite side, there is a corresponding asymmetry in the mouth and the position of the ears. As the child nears the period of adolescence with the deformity uncorrected, a definite rotary lateral curvature of structural character is likely to develop in the cervical segment of the spine, its convexity toward the normal side, and still later a compensatory curve appears in the dorsal segment with its convexity in the opposite direction. Occasionally, and especially toward the epoch of adolescence, this compensatory curve may assume a high degree of distortion.

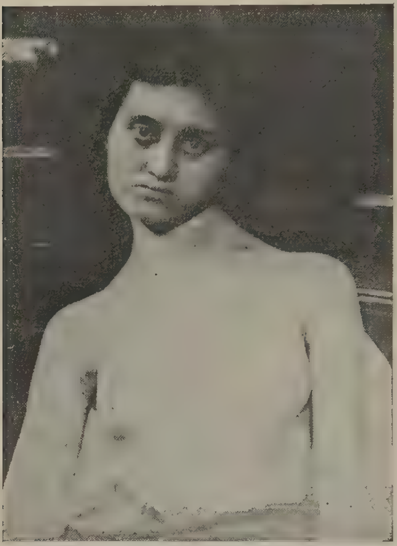


Fig. 165.—Wry-neck as the result of tuberculous disease of the cervical spine in childhood.

Differential Diagnosis.—The point of greatest importance here is to distinguish between the congenital forms of torticollis, whether of muscular or osseous origin, and the acquired forms in which the abnormal position of the head represents the expression of disease. The most frequent and most important of such conditions is tuberculous disease of the cervical spine. Especially in such involvement of one atlanto-occipital joint do we find a position of the head closely simulating that of congenital torticollis; this may be also true, however, of tuberculosis of the cervical segment lower down and particularly if the disease is situated with preponderance toward the one side or other. This is often the case in the upper four cervical vertebræ, presumably because of the small size of the bodies in this region. The distinguishing mark of tuberculous disease of all joints will help us here also, namely, limitation of motion to some extent *in every direction* by reason of muscle spasm. In congenital muscular torticollis, motion in the direction of still further shortening the short sternomastoid can be made without hindrance; in joint disease this is not so. Resort to the *x*-ray and the dermal reactions to tuberculin (Moro, von Pirquet or intradermal injection) will assist in clearing the situation.

Acute, or Rheumatic Torticollis.—This is to be looked upon as an acute myositis; it has a sudden onset, usually accompanied by fever and the other signs of mild general infection. It is usually spoken of as "stiff neck" and should by reason of its onset and general nature cause no diagnostic difficulty in this connection. There is usually pain produced by attempting to overcome the distortion with the hand and there is most often some tenderness on palpation of the contracted muscles.

Reflex Torticollis.—While the torticollis of tuberculous disease is to be looked upon as deserving this name also, other conditions of more acute character may produce a distortion of similar appearance. Among the conditions thus to be considered are tonsillar and peritonsillar infections, adenitis, dental infections and trauma of degrees more or less severe.

Spasmodic Torticollis.—This is a condition very rarely seen in childhood. As its name implies it is an asymmetrical position of the head produced by spasmodic muscular action of either clonic or tonic character. The deformity can usually be temporarily corrected by the hand although it at once recurs when the hand is removed. When seen in children it is usually to be regarded as a psychoneurosis.

Prognosis.—The prognosis of torticollis depends upon its cause. Congenital torticollis is a condition which can be completely cured by proper treatment, so long as the opportunity for this be given before secondary changes of structural nature have assumed a high degree; therefore before the child is well advanced into adolescence. Even then and later the torticollis is most often susceptible of complete correction; a long time must elapse, however, before the facial asymmetry will disappear. Immediately after correction of the torticollis, in any case, the facial deformity is likely to appear exaggerated; very often parents are so accustomed to the asymmetrical position of the head in older children, that they are entirely unaware of the facial deformity until their attention has been called to it. When structural scoliosis is present below the cervical segment, it will require subsequent and extended treatment on its own account and its prognosis is that of this condition generally and by no means favorable as to complete cure.

Regarding the acquired forms of torticollis merely as symptoms of the abnormal processes underlying them, nothing further need be said on this score in this place.

Treatment.—At the present time, the operative treatment is the only one which should be considered for cases of congenital torticollis. Formerly mechanical methods were in vogue, particularly for the milder degrees of deformity and in young children. Such treatment has now been discarded for the reason that complete cure rarely resulted, that much time and effort were required together with the wearing of unsightly apparatus and because only too often after a time it was realized that operation must be done if a satisfactory appearance was to be secured. In young infants torticollis of moderate degree may be overcome by very persistent methodical stretching of the short sternomastoid muscle. As a rule the children are brought for treatment at too late an age for this however; the usual indication when they come is for operation by incision of the short structures.

Tenotomy.—The operation which is usually required is the division of the sternal and clavicular insertions of the sternomastoid muscle. In pre-antiseptic times this was accomplished by subcutaneous tenotomy, for obvious reasons. By Lorenz and certain others this procedure is favored, even now, in order to avoid the disfigurement by the scar necessarily resulting from open incision. If the operation be properly done, the scar is so small and so placed as to be unobjectionable. On the other hand, with aseptic methods efficiently carried out, the risk involved is greater in the subcutaneous method of operation by reason of the possible wounding of the internal jugular vein which lies just mesial to the mesial border of the sternal head of the muscle. One is also less likely to be able to divide thoroughly all of the fibrous bands in the subcutaneous procedure.

Technic.—The patient is placed with the shoulders resting upon a sand-bag, the head being allowed to fall backward, throwing the sternomastoid into strong relief. An incision is made, following accurately the folds of the skin, between the sternal and clavicular insertions, just above* the clavicle and varying in length from ¾ to 1½ in. in length according to the child's size. The sternal tendon is divided upon a director which has been passed under it and this is followed by the division in similar manner of the clavicular tendon, as well as any other resistant bands of tissue. A very careful suture of the skin is then done, preferably by a subcuticular method or by the application of the Michel clamps, so as to secure the most accurate coaptation possible. A small aseptic dressing is applied to the wound. The operator now takes the patient's head in his hands and, with great care, secures the maximum degree of over-correction by turning the head so that the chin points to the side of the shortened sternomastoid and by bending the neck so that the ear of the normal side approaches the shoulder of that same side. If this redressement be thoroughly done, one can usually perceive the yielding of fibrous strands. The head is now held by an assistant in the over-corrected position while the dressing is applied.

In the application of this dressing, the practice of different operators varies considerably. Many men of experience utilize a plaster-of-paris dressing which envelopes the head, holding it well over-corrected, and the whole thorax. The writer has not used this dressing for a number of years, applying instead a very voluminous dressing of cotton wadding and gauze bandages enveloping only the neck, after the method of Schanz. If the dressing be made extremely heavy on the side of the operation, and if the whole dressing be made quite firm, the retention in the position of over-correction will be quite as satisfactory as with plaster; on the other hand, it is both more comfortable to the patient and easier to apply. That which is indispensable if a good result is to be obtained and which makes unnecessary the elaborate after-treatment described by some authors, is the thorough over-correction after the muscle has been divided. The dressing, whether of plaster or of wadding, should be retained for a period of from four to eight weeks, depending upon the age of the child and the degree of deformity. For several weeks after its removal it is advisable to make daily manipulations in the direction of over-correction, so as to stretch the formerly short muscle to the utmost. In older patients, with a degree of structural scoliosis already developed, supplementary treatment will be required in the way of corrective manipulations, gymnastics and massage; corrective plaster jackets may be necessary as preliminary to these.

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* During the past few years the writer has been making the incision just *below* the clavicle and sliding the upper lip of the wound upwards. The scar is thus made least conspicuous.

Inasmuch as the incision of the sternomastoid at its lower extremity, as above described, usually leaves the muscle of the operated side less prominent than that of the other, Lange prefers cutting the sternomastoid close to the mastoid process rather than at its sternal and clavicular attachments. The incision is made along the posterior border of the muscle and care must be taken not to injure the spinal accessory nerve and the internal jugular vein. This is to be accomplished by making the division high up and by blunt isolation of the muscle upon a director before incising it.

It cannot be too strongly emphasized that the earlier the operation of congenital torticollis is done, the better will be the result and the less attention to secondary deformities will be required, whether of the face or of the spine lower down.

The discussion of the treatment of the acquired forms of torticollis will not be undertaken here since it depends upon the primary causative condition, of which it is but a symptom.

CURVATURES OF THE SPINE

Anatomical Considerations.—It is perhaps unfortunate that we ever use the term "Curvature of the Spine," for the reason that every spine is curved and because in certain abnormal conditions we are dealing with exaggerations of the normal curves, whereas at other times we have to do with curvatures in entirely different sense; moreover, we often find conditions termed curvatures in which we are dealing simply with postural faults, whereas in other cases, also spoken of as curvatures, we shall be concerned with structural changes and even with the results of disease actively destructive in character. It were much better then for us to leave the term "curvature" of this or that character for the layman to use and to confine ourselves, as physicians, to the use of designations which are in accord with what we know of the pathology of the conditions in question.

Physiological Curves of the Spine.—It is customary to refer to these as the curves in the sagittal or anteroposterior plane of the body which we recognize as normal in the fully developed individual. We know that the cervical segment has its convexity forward, the dorsal segment its convexity backward and the lumbar its convexity forward. We speak of the curvature with convexity forward as lordosis; that with the convexity backward, as kyphosis. Whereas these terms were used originally for abnormal conditions only, they are now used by some writers as designations for the normal curves also. During the first three or four months of life there is normally only one of the physiological curves to be seen; this is the curve backwards and it may be recognized even though in lesser degree, in the fetus. The other curves appear with the assumption of greater physical activity; after the fourth or fifth month, we may usually recognize the cervical curve forward and by the end of the first year of life the forward curve of the lumbar region, the lumbar lordosis, has become apparent.* This corresponds with the development of the function of standing. Once the erect position has been assumed, we can understand that the physiological curves become interdependent; the dorsal kyphosis will depend for its degree to no small extent upon the degree of the lumbar lordosis and the cervical lordosis or forward bend, upon the degree of dorsal backward bend. By as much as these curves are reciprocal in this manner, they are spoken of as "compensatory." There is as yet no generally approved norm for the degrees of curvature in the different segments nor in their relation to one another. This is largely because there is necessarily a considerably wide margin of variation from individual to individual without transcending the limits of the normal and depending largely upon personal height and peculiarity of build. According to Lovett,† one simple relation seems fairly constant above the age of 10 or 12; a plumb line held against the back of the sacrum touches or comes near the convexity of the dorsal spine and in young children it cuts this convexity.

It must be apparent that the anteroposterior curvatures of the spine, since they are to a large extent interdependent, must stand in some relation to what is known as the inclination of the pelvis. As Lange has shown by simple diagrams (*loc. cit.*) an increase of the inclination of the pelvis, as indicated by the lowering of the front and raising of the back, must be accompanied by an increase of the lumbar lordosis and *vice versa.* What determines the variations in pelvic inclination in different persons seems to be but little understood.

Lovett‡ has devised an apparatus by which the relations of the anteroposterior curves of the spine and their relation to the general posture of the body may be graphically expressed with accuracy. Even by this means, however, no absolute norm for posture has as yet been agreed upon. At the same time there is general understanding as to what constitutes good and bad posture; it is apparent even to the eye of the layman and indeed it is by him that most of the abnormal postures are discovered and brought to professional attention, in the first place.

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* Lange, in Pfaundler u. Schlossmann, Handb. d. Kinderheilk., Bd. v (1915).

† "Lateral curvature of the spine and round shoulders" (1907), p. 167.

‡ *Loc. cit.*, 3rd ed. (1916).

As far as deviations to either side, from the perpendicular, are concerned, there is a common notion among people that no one has a perfectly straight spine. For this reason, it is not uncommon to observe unconcern expressed when the diagnosis of lesser degrees of lateral deviation of the spine has been made. As a matter of fact, most authors describe as physiological a slight deviation toward the right side. It has variously been ascribed to righthandedness and to the pressure of the aorta. However this may be, in children at least, a lateral deviation sufficiently pronounced to be made easily apparent to a lay eye, is to be regarded as abnormal even though not necessarily of serious import. The physiological deviation toward the right is described as usually found in the lower half of the dorsal and the upper portion of the lumbar segment.

Movements of the Normal Spine.—The most comprehensive study of the movements of the normal spine, is that of R. W. Lovett.* To consider this in detail would carry us too far for our present purpose. The freedom of movement in every direction in the spine of the child is relatively greater than in the adult but in a general way the mobility of the three segments corresponds as regards the relative degrees; the dorsal segment is the least mobile of the three as regards movement in the anterposterior plane, whereas the movement of twisting or rotation is here of greater amplitude than in the lumbar segment.

It is important to remember that in the position of forward bending, as in touching the toes with the fingers while sitting with the legs completely extended, the spine should make a uniform and unbroken curve from the sacrum to the root of the neck.

Anatomical Landmarks.—Whereas the seventh cervical vertebra is called "prominens" because of its long spinous process, that of the first dorsal is usually more prominent. The spine of the fourth lumbar vertebra corresponds in level to the highest point of the iliac crests; it is thus easily identified by the line joining them and it is the point most commonly used from which to count in the attempt to locate a particular vertebra. While one may count from the seventh cervical for this same purpose, it is not always easy to avoid confusing the seventh cervical with the first dorsal for the reason above mentioned. It is well also to remember that the inferior angle of the scapula should correspond with the spinous process of the seventh dorsal and the root of the scapular spine with the spinous process of the third dorsal.

DEVIATIONS OF THE SPINAL COLUMN IN THE ANTEROPOSTERIOR PLANE

The Round Back.—An exaggeration of the backward curve which is normally present in the dorsal segment of the spine may be found as the expression of various abnormal conditions. It is spoken of generically as kyphosis; a term which requires to be further qualified by a designation indicative of the underlying or causative condition. Thus, kyphosis results commonly from the destruction of vertebral bodies by tuberculous disease, in which case we speak of "angular kyphosis," or better, of tuberculous kyphosis. Similarly, as the result of the softening of the bones produced by rickets and osteomalacia, kyphosis is often encountered and must be spoken of in accordance therewith. On the other hand, a large group of cases is observed in which kyphosis exists, not as the result of a pathological process which has produced an alteration in the physical characteristics of the spinal bones, but which must be looked upon primarily as a fault of attitude or posture. Leaving the discussion of tuberculous deformities of the spine for the chapters devoted to that subject, it is desirable to apply ourselves to a description in some detail of the rachitic and purely attitudinal kyphoses because of their frequency and importance during the age of childhood.

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* *Loc. cit.*

Rachitic Kyphosis.—The rachitic kyphosis is one of the frequently observed deformities accompanying the disease so well known as Rickets; a disease whose characteristic effects upon the structure of the bones is described elsewhere in this work. Suffice it, in this place, to say that rickets produces a softened condition of the bones during the phase of its activity as the result of which they yield in an abnormal manner to superincumbent weight and to muscular stresses, thus giving rise to the various well-known deformities, such as bowlegs. Rachitic kyphosis is one of the typical deformities seen in early childhood and during the activity of the disease. The most usual situation of the backward bend in the spine, in this condition is in the region of the lumbodorsal junction. In its most characteristic form, it involves the lumbar segment which shows not only a loss of the forward, or lordotic bend which is normally here found, but now displays a decided prominence backward. It is seen to best advantage as the child sits on its bed, with the knees fully extended, the hips flexed at a right angle. Indeed, according to Lange,* we find in this position the most plausible explanation of the causation of this deformity. The tension of the ham-string muscles serves to tilt the lower part of the pelvis forward, necessarily causing the lumbar segment of the spine to bow backwards, by reason of the very limited amount of motion which is possible at the lumbosacral joint in the anteroposterior plane.

The **diagnosis** of this deformity is usually quite apparent upon careful examination, in view of the other symptoms of the disease which are uniformly present. It is to be differentiated from the tuberculous kyphosis by reason of the larger number of vertebræ which are affected in rickets and by recognition of the marked stiffness of the spine in tuberculous disease which is produced by muscular spasm.

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* *Loc. cit.*

A degree of stiffness is often found in the rachitic spine; it is associated however, chiefly with motion in the anteroposterior plane. If the child be placed upon its abdomen, the chest applied to the examining table, and the body be raised by means of the feet in the hands of the examiner the normal spine should make a uniform curve with its convexity forward; there should be free mobility of the lumbar segment as the legs are swayed from side to side, in this position. In tuberculous disease there is marked limitation of this lateral mobility of the lumbar segment, as well as that in the anteroposterior plane. In conjunction with the evidence of destructive disease to be seen in the *x*-ray plate we have here the means of satisfactory differentiation.

The **prognosis** of rachitic kyphosis is excellent provided that it is recognized early and given the proper treatment, both as regards the spine itself and the general management of the constitutional condition. Unfortunately, it is too frequently regarded lightly and left for a spontaneous recovery which does not take place. In this event, as has been emphasized by many authors, it frequently serves as the foundation for other structural deformity of the spine, chiefly scoliosis, which develops upon this basis at the time of adolescence or even much earlier than this. This is ground for giving to rachitic kyphosis serious attention, beyond the constitutional measures commonly employed.

The **local treatment** of rachitic kyphosis should be undertaken while the disease is still active if it is to produce its best results. Still more important however, is the prevention of deformity of the spine in the rickety infant. While the disease is active, the child should not be encouraged to sit on its bed, as is frequently done by mothers; when the back has become strong it will be difficult to restrain it from doing so. The bed upon which the child lies should be firm and not yielding. In giving the child the rubbing, so commonly employed in rickets, the back should not be neglected.

Once the tendency toward kyphosis has become evident, or in case it has developed to a degree, energetic measures are indicated with a view to establishing a definite lumbar lordosis while the bones are still in their abnormally soft condition. The recumbent posture is imperative if this is to be accomplished and means must be employed to keep the lumbar spine curved forward continuously. It is common to employ the Bradford gas-pipe frame for this purpose. Folded towels placed along either side of the spine are used frequently to hold it forward; a better method is to use a swathe of ticking about the lower part of the trunk, on which a cylindrical cushion stuffed with cotton has been sewed on either side of the spine. This does well enough in hospitals where expert nursing care is available. For use in the home and, for that matter in hospital also, a very much better method is found in the application of the so-called plaster bed. This is a shell of plaster of paris which is molded to the body while it is held on a suitable frame, or otherwise, in a distinctly lordotic position.

When properly padded, it is perfectly comfortable; it ensures the continuous preservation of the desired posture and it has the advantage that the child may be carried about without disturbing this. In addition, the child should receive daily massage of the back muscles and when the back has become so far recovered that the child insists on sitting up, it should be given exercises to strengthen the spinal muscles. These may be given by placing the child on its abdomen and encouraging it to play with toys held above it, so that the lumbar muscles come into active play. Only by systematic employment of such measures can the complete recovery be assured.

Postural Kyphosis.—This condition is otherwise spoken of as faulty attitude, round shoulders, stoop shoulders and round back. Whereas, it appears as a rule during the school age, many of the more severe cases will be found in children who have had rickets and to which disease it may be regarded as bearing a vestigial relation. In its usual form this is, however, not the case; the deformity or faulty bearing involves the dorsal segment, for the most part, rather than the dorsolumbar. Most often the child is one of less than ordinary vigor and there are often associated other postural faults, such as weak or flat feet and knock knees. While the abnormally great backward convexity of the dorsal spine is the most striking characteristic, there are usually other features indicating the faulty carriage of the trunk. The head is carried forward, the shoulders droop, the chest is flat and the scapulæ are prominent, so that they have been spoken of as "winged" or as "angel wings." The lumbar spine is usually more lordotic than normal and this has been spoken of as a "compensatory" condition; it is not always the case, however, being sometimes of normal degree and in some cases flatter than normal. The latter condition is apt to be found in children who have been rachitic, whereas the hollow round back would seem to stand in some relationship with an unusually high degree of inclination of the pelvis, whatever the cause of this may be.

In its essence, postural kyphosis is to be regarded as the result of muscular insufficiency; the erector spinæ muscles being unequal to the task of maintaining a proper posture, the child seeks to throw itself upon its ligaments for support. The posterior ligaments are not put upon the stretch, however, until a considerable augmentation of the normal backward bend of the dorsal segment has been attained. As a rule, the kyphosis remains entirely flexible and disappears in the recumbent position. Sometimes, and especially in the presence of marked deformity, stiffness develops and these cases have been spoken of as "resistant round shoulders." It should not be overlooked, however, that in quite a few instances this fault of posture must be regarded as hereditary, as a family characteristic; this is particularly likely to be the case with the kyphoses situated high in the dorsal segment, beginning with the first or second dorsal vertebra. In the experience of the writer this fact is of important prognostic significance;

these cases do not respond to treatment nearly so well as those which are obviously of purely muscular origin and they call for much more intensive and prolonged care. The prognosis of round back or round shoulders thus depends not so much upon the degree of backward curvature, as upon its nature and position. Under the most favorable mechanical conditions, however, the prognosis must rest not upon the application of some form of support, but upon our ability to develop the necessary amount of muscular strength for the child. Very frequently this fault of posture is looked upon as evidence of carelessness in the child and parents believe that it could overcome the fault by persistent endeavor. This is doubtless true, sometimes; usually this is not so. The faulty posture is to be regarded as due to the loss of that reserve supply of strength upon which the normal posture depends and by reason of which it is maintained automatically and without special effort of the will. Our endeavor must be to restore that automatism. On the other hand, the mental attitude of the patient must be taken into account very carefully if we wish to form an accurate prognosis in any case. Unless we can secure a spirit of coöperation on the part of the patient, our efforts at the correction of postural kyphosis are not likely to meet with success. It is unfortunate that the correction of this condition is very often regarded as very easy and requiring at most a certain amount of "gymnastics" of ever so perfunctory a kind; this is by no means the case. Most children object to the interference which the proper treatment causes with their leisure and play; if they must receive their first lessons in obedience as a part of the treatment for faulty attitude, failure is apt to result on both accounts.

Treatment.—Before proceeding with a discussion of the treatment to be pursued in cases of fully developed postural kyphosis, something further had best be said concerning some of the contributory causative factors, knowledge of which is important both for the purpose of prophylaxis and in order to make more effective the therapeutic measures to be recommended. The condition of muscular weakness which is so important a factor may be ascribed to bad hygienic conditions in the home, exhausting illness, overwork, psychic disinclination to healthful exercise and the rapid growth which is incidental to the epoch of adolescence. The mechanical factors which lead to an habitual flexed position of the weak spine are of great importance in the etiology. They embrace refractive errors of the eyes, necessitating a close reading distance, improper furniture in school or home and the drag upon the shoulders made by suspending the clothing from them. The rôle of this last factor is commonly overlooked. The clothing should be suspended from a belt or from a waist so constructed that the pull comes from points close to the neck rather than from the shoulders themselves, as is so commonly the case.* These factors are of importance not only in preventing faulty postures, but they require to be taken into account if we are to succeed in treatment when they have developed.

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* Goldthwait, Am. Journ. Orth. Surg. (1903), p. 64 *et seq.*

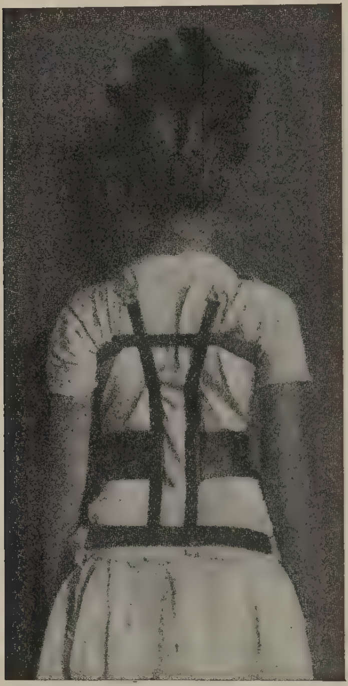

Fig. 166.—Round shoulder brace. (Goldthwait.)

We have but one method open to us for combating the element of muscular weakness wherever we find it operative in disturbing normal function and that is by the cultivation of strength by suitable and properly regulated exercise. The use of supports, far from aiding us by maintaining an improved posture, does just the reverse by interfering with movement and the action of the muscles associated therewith. Supports will in some cases be necessary for a time, nevertheless, because of excessive weakness or unusual degree of deformity, they should not be employed as a substitute for postural training and gymnastic treatment, however much they may be demanded by parents as constituting an easier and more convenient method. While the support may be made to improve the appearance while it is being worn, there must come a time for discarding it and the muscles will then be found not to have grown stronger under its use but rather the reverse, unless they have been made stronger by gymnastic methods. Once it has been determined that mechanical support is necessary, it should be effective and had best be made of tempered steel and properly fitted. The round shoulder brace of Goldthwait* is one of the most satisfactory (Fig. 166). It should be worn constantly except during recumbency and while gymnastic treatment is being given.

Gymnastic Treatment.—This treatment is of real effectiveness only when it is individualized and carried out in a purposeful manner. There is but little to be expected of the advice so often given, that the child should have gymnastics, and as the result of which it is placed in a class containing normal children, scoliotics and others with postural faults of different kinds. To obtain results of satisfactory character, the treatment must be suitable for the individual child both as concerns the amount and the kind of exercises and corrective manipulations; for the most part the assistance of the gymnast is required in each exercise in order that it may have the desired effect and therefore but one patient may be treated at one time by one person.

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* *Loc. cit.*

To discuss the gymnastic methods in detail would carry us too far for the present purpose. For this reference should be had to special work on this subject. It will be enough to say here that there are two distinct parts to this form of therapy. It is necessary first of all, to enable the child to assume a correct position. In order to accomplish this, it is often necessary to stretch the soft tissues of the anterior aspect of the spine and trunk, whose shortened condition accounts for whatever stiffness may be present. In the second place it is necessary to teach the child how to carry itself correctly, because it has lost the sense of correct posture to greater or lesser degree. Finally, exercises must be chosen which will develop those muscles whose function it is to maintain the proper posture and whose insufficiency is expressed in the postural fault. If the choice is appropriate and the execution is correct, the exercises will themselves produce postural correction while they are being done. Effective gymnastic treatment should, furthermore, result in gain of weight and an obvious improvement of the general condition. Should the opposite be the case, it may be assumed that the treatment requires to be modified for the patient in question or that there may exist some condition which contraindicates what is being done. A careful examination of the heart, lungs and other important viscera should be made as a preliminary to treatment, in any event; by this means such contra-indications may practically always be forestalled.

No discussion of the treatment of postural faults of the spine would be complete without mention of the great value of massage, in connection with the gymnastic method. It is employed with great advantage at the conclusion of each gymnastic treatment and it furnishes a means of improving the vigor of the spinal muscles which is distinctly worth while. Its value must be spoken of in this connection with two reservations, however; it is not of much use as an independent method and to the exclusion of gymnastics and it is not to be confused with the unmethodical rubbing or the so-called "adjustments" which are now so often employed by untrained persons calling themselves masseurs, on the one hand, or by the different kinds of irregular practitioners and "paths," on the other. Massage is a system of manual treatment of the soft parts, requiring accurate anatomical knowledge, training and skill; it accomplishes changes in the nutrition of the accessible parts of the body chiefly by its effect upon the circulation of blood and lymph. It accomplishes no miracles, however useful it may be. Although abundant opportunity has been given during the past decade for observing the effects of the different kinds of "spinal adjustments," because of the considerable vogue which they have had in this country, there is no reliable evidence to show that they are to be credited with any value in the correction of tangible physical defects of the spine or that it is even justifiable to speak of them as being entitled to comparison with massage; there is certainly no reason to speak of them as being in any sense identical with massage nor can they be offered as satisfactory substitutes for it when a real indication for massage is present.

Just as the writer has refrained from discussing the details of the gymnastic treatment to be employed in postural kyphosis, as not lending themselves to a work of this kind and extent, so and for the same reason, the matter of measurement and record of the deformity has been merely mentioned. This is not because it is thought to be of minor importance; the reverse is true. It is truly essential that such record be kept if we would have anything like an accurate conception of what is being accomplished. However, up to the present writing, there is no general agreement concerning the method to be employed for this purpose and the various forms of apparatus which have been proposed are of such character that they require specialized training in their use; moreover, they are not likely to be found in the hands of those who do not specialize in orthopedic surgery.

Lordosis.—An increase of the normal forward curve of the lumbar, or sacrolumbar region, which is spoken of as lordosis, is very frequently seen in childhood. Whereas kyphosis is entitled to consideration as a condition requiring attention on its own account, this is not so with lordosis; this is because abnormal lordosis is almost always symptomatic of some other pathological condition, requiring separate recognition. Because of their importance in diagnosis, mention is made of the principal conditions which may be productive of the lordotic posture without entering into a detailed discussion in this place. They may be thus classified:

(*A*) Conditions associated with increase of pelvic inclination.
  1. Flexion ankylosis or contracture of the hip, from tuberculous or other disease of this joint.
  2. Congenital Luxation of the hip.

(*B*) Conditions of the Spine itself:
  1. Early stage of Lumbar Tuberculous Spondylitis.
  2. Spondylolisthesis.
  3. Dystrophic and Paralytic Affections.
  4. Rachitic disease.

In view of what has been said above, it will be apparent that Lordosis does not require treatment directed to it, but rather toward its cause; the same thing may be said of lordosis in its relation to diagnosis and prognosis.

Spondylolisthesis.—This is an unusual condition under any circumstances and is most rare before the age of puberty. I have never seen a case except in an adult. For this reason it does not seem advisable to devote space to it in this work (see Malformation of the Spine).

LATERAL CURVATURE OF THE SPINE (SCOLIOSIS)

When an individual presents a *persistent* deviation of the spine, in whole or in part, to either side of the median line we speak of lateral curvature or scoliosis. It is, however, necessary at the outset to distinguish between what may be termed the scoliotic posture and scoliosis of essential character. Since the normal spine is flexible to a high degree, every one may and does assume scoliotic postures at times; since these postures are not persistent and merely incidental to normal function they require no further mention. When an individual with a short leg stands on both feet with the shortening uncorrected, it is apparent that the pelvis will be tilted and that there will be a deviation of the lumbar spine with its convexity directed toward the side of the short limb. When the shortening is corrected by placing a support of the necessary height under the short limb, or when the individual is examined lying in the prone position, the deviation of the spine disappears entirely unless the spine is itself abnormal. This is true even though he has walked for years with the difference in length uncorrected, provided that the asymmetry is not of high degree and that the spine is itself normal; it is a matter of very common observation in persons with unilateral congenital luxation of the hip, with limb shortened by fracture or by other causes. Under such circumstances we speak, not of lateral curvature or scoliosis but rather of a scoliotic posture or of incidental scoliosis. On the other hand, we find certain cases in which there is no fundamental asymmetry of the lower extremities or the pelvis and in which the spine persistently deviates toward one side or the other but in which the deviation disappears when the patient lies prone upon a flat surface; the further examination fails to develop any structural change of the spine or thorax. The curvature is, however, constantly present when the patient is standing and we, therefore, speak of it as scoliosis; this is however, postural as distinguished from structural scoliosis in which there is a residual deformity of spine or thorax of varying degree which does not entirely disappear in any position which is assumed. The two conditions would, on this account, appear to be fundamentally different. It is easily conceivable that the postural form might be causative of the structural form; this relation is credited by some writers but by others it is denied. It is not to be doubted, as a matter of common experience, that postural scoliosis may exist for many years without the development of the structural changes characteristic of structural scoliosis. Furthermore, it is commonly noted that structural scoliosis appears in individuals subject to careful though ordinary observation and in whom no pre-existing postural fault has been seen. In such patients the first examination reveals the fact that although the deformity may be very slight, undeniable evidence is found that structural changes have already taken place in the spine and the thorax. These considerations make it seem desirable that postural scoliosis should be placed under a rubric separate from that assigned to the structural form. This is emphasized by the fact that the two conditions must be looked upon as entirely different in their clinical features, in their prognosis and in the methods of treatment to be adopted.

Postural Scoliosis.—This form of scoliosis is also called "functional" and because it so often appears coincidental with the early years of school attendance, it is often spoken of as school scoliosis. Lorenz has indeed spoken of it, on this account, as the "occupational deformity of school children." Because in the majority of cases the deviation consists of a long curve of the spine extending from neck to sacrum, toward one side or the other, it is also known as "total" scoliosis. It is estimated that the curvature is found with its convexity to the left in about 90 per cent. of the cases. In the experience of the writer, postural scoliosis is, however, by no means always "total" in its extent. In some cases it is confined to the dorsal segment and is then likely to be associated with round shoulders or postural kyphosis. In other instances it may be found chiefly in the lumbar segment. It must be said, however, that under these circumstances it is apt to be found as the forerunner of a truly structural scoliosis. Whereas structural scoliosis is observed much more often in girls than in boys, the reverse is said to be true with respect to the purely postural form. According to Lovett, total scoliosis is found between the ages of 5 and 18 years, as a rule.

The milder forms of postural curve are by no means easily recognized by the untrained, unaided eye and it is always best to carefully mark the tips of the spinous processes with a skin pencil; after this has been done, a plumb line may be held so as to cut through the cleft of the nates and the deviation of the line of spinous processes may then be accurately with this as a guide. The method of making a permanent record of this deviation will be described in connection with structural scoliosis (*q.v.*, p. 340). When the patient bends forward with the trunk at a right angle to the thighs and the back is observed from the rear of the body, there is either no deformity of the chest wall to be seen or else the chest appears prominent on the side of the *concavity* of the curve. Inasmuch as in structural scoliosis the thoracic wall is higher on the side of the convexity, due to the torsion of the spine which carries the ribs with it, this prominence of the chest on the concave side in postural scoliosis is sometimes spoken of as "paradoxical torsion deformity." Its cause has been the subject of much discussion but it is explained by Lovett as a physiological phenomenon and due to the fact that "a spine making any bend convex to the left in the erect position will turn at its upper end to the right." He says further: "The thorax and shoulders will be twisted backwards on the right, and when the patient bends forward, this twisted position of the shoulders may be carried over into the position of forward bending if the case has been of long standing and the right side of the back will be higher in this position."

In addition to the left convex curve, these patients carry the left shoulder high and forward, the right shoulder low and backward. Whether a pronounced kyphosis be present or not, these patients have a bad carriage; the chest is flat and the abdomen is likely to be more prominent than normal.

The cause of postural scoliosis must be sought, as in postural kyphosis, first of all in muscular insufficiency together with the action of such factors as make likely the frequent or habitual assumption of asymmetrical positions of the body. For this reason, bad seating and working conditions at school have had an important etiological character ascribed to them. This would seem to be especially appropriate in association with constitutional enfeeblement, long continued tasks in the sitting position and abnormal conditions of the eyes which cause the head to be habitually held to one side. The slant of the usual script, in writing, has had much causative importance attributed to it; in the writer's opinion without much reason. Through the work of the Boston Schoolhouse Commission much stress has been laid on proper school furniture in the prevention of scoliosis and their observations would appear to have especial importance with respect to the postural variety. Largely as a result of their work there has come about a most useful reform in this regard.

In consideration of what has been said of the nature of postural scoliosis the diagnosis will appear to be associated with no particular difficulties. At the same time, it may be profitable to call attention to certain errors which are often made. A lateral curvature sometimes appears to be postural when it is really representative of an early phase of structural curvature. Usually, it is possible, even in the earliest stages of structural curvature, to ascertain the existence of structural change; when the patient is examined in the forward bending position, or better still, lying prone on a perfectly plane hard surface, it is usually not difficult to appreciate the persistence of deformity and especially as concerns the torsion or rotation of the spine toward the side of the convexity. Occasionally however, one sees patients so early in the course of the deformity that error is made because of the very great flexibility of the spine which still exists. Under such circumstances and until the real nature of the deformity becomes apparent by the appearance of unmistakable structural change, diagnostic error would seem excusable, for the time being, at least. Sometimes, lateral postural curvature is not recognized because the spine is not examined with sufficient care; the case is looked upon simply as one of bad carriage or round shoulders for this reason. Not infrequently the erroneous diagnosis of postural scoliosis is made when one is really dealing with a symptomatic scoliotic posture; the patient has a short leg which causes this by tilting the pelvis. It should always be ascertained whether the pelvis is level or not and whether the limbs are of equal length. The need for emphasizing this precaution lies in the fact that although most of the instances of asymmetry in the length of the lower extremities have their cause in conditions which are quite striking in their other clinical characteristics, as for example congenital luxation of the hip, tuberculosis of that joint, infantile paralysis, there are many other cases in which the shortening as well as its cause, is not easily apparent. The shortening may be congenital and without other explanation; a congenital difference of ¼ in. in the limbs of a well grown adolescent is far from uncommon, even though it is scarcely enough to produce a scoliotic posture. More than this may well do so, however, and the shortening is always important to recognize; even if insufficient to be considered the real cause of the deviation, it may be the factor which has determined the lateral bend of the spine in association with an insufficient musculature. It must therefore be taken into account in connection with the treatment. Recognition of shortening of considerable degree should always demand careful investigation of its cause. Sometimes this may be found in the hip joint; depression of the neck of the femur, coxa vara, is not seldom the cause. It is to be recognized by the position of the great trochanter above Nelaton's line, the head of the femur being felt in its normal place under the pulsation of the femoral in the groin, by the limitation of abduction and internal rotation of the hip and particularly by the definite information of the *x*-ray plate. While coxa vara is usually bilateral, it is occasionally unilateral or else so different in degree on the two sides as to account for a distinct spinal deviation to one side. Marked weakness of one foot in the sense of decided planovalgus deformity, has been regarded by some as the determining factor in postural lateral deviation of the spine; it is frequently present in these patients, as might be expected, since it is itself to be looked upon as an expression of muscular insufficiency and by that token is itself a postural deformity.

There remains to be mentioned, as of importance in the differential diagnosis of postural scoliosis, the scoliotic posture as a symptom of hysteria. This is seen, not infrequently, in children as well as adults. It is invariably found as a rather sharp bend at the sacrolumbar junction of the spine, the dorsal segment being quite straight or deviated slightly to the opposite side. In prone lying or in suspension from the head, the deformity disappears entirely. Most often some pain is complained of; usually it is localized in the region of the hip of the side of the concavity. The well known general characteristics of hysteria are present in these patients and there is occasional association with hysterical contractures of joints of the extremities, most often the foot of the side of the spinal concavity. In two cases which the writer has seen, the onset was sudden and associated with emotional disturbance.*

It is a notion with many physicians, and of course with more laymen, that lateral curvature is likely to be "outgrown" as the years go by and that it is not to be regarded as requiring treatment. There is no evidence to show that postural scoliosis ever disappears spontaneously; it will not do so under approved methods of treatment unless they are very thoroughly carried out. In the case of the structural variety, we shall later see that it is altogether out of the question. On

* Hovorka, Zeit. f. Orthop. Chir, Bd. xiv (1905).

the other hand, may we not regard postural curvature as an innocuous cosmetic fault whose correction is bound up with much inconvenience, which is likely to involve no danger to the patient and which therefore need not necessarily be given treatment? There are three good reasons why we should say no to this question. The existence of postural scoliosis presupposes a condition of unsatisfactory muscular control which is itself legitimate ground for attack in the interest of the patient; it is likely to bespeak the existence of improper and unhygienic conditions at home or at school whose correction is a necessary part of the treatment. Postural scoliosis may be the forerunner of structural changes in the spine, whose limits cannot be foretold, which may be impossible of complete correction and which may constitute a cause for shortening the expectancy of life. There is good reason to believe that uncorrected postural scoliosis may be the cause of very intractable backache later in life, especially in women. On the other hand, if given adequate treatment, scoliosis of postural character may be completely and permanently cured.

Treatment.—In the first place, this calls for the correction of such unsatisfactory unhygienic conditions as may be found to exist, whether they concern the general health or the posture itself; whether they are to be sought in the home or in the school. The conditions in question which may require attention are of the same general character as those which come for consideration in connection with postural kyphosis. The state of general nutrition is of great importance and it may call for careful direction as regards the diet; under-nourishment is frequent in these cases, even among the children of well to do parents. In this case it is apt to depend upon improper food and often upon the inordinate consumption of sweets. It has been sought by some to connect the existence of postural faults with obstructive conditions in the upper respiratory tract, such as adenoids and diseased tonsils. If there is such a connection, it is probably of quite indirect character but attention should undoubtedly be paid to it. Many of these children are indisposed, by taste, to outdoor sport and exercise; they prefer to spend their leisure in reading and are likely to be found reading in very bad sitting positions at home, as well as at school. The influence of improper suspension of the clothes upon the carriage of the child has been referred to in connection with postural kyphosis and what has been there said may with equal fitness be repeated with respect to postural scoliosis. Regard must be had for this whole group of personal factors if we wish to meet with the utmost success in treatment. Of very great importance is the correction of ocular defects which may exist; without this it may be impossible to prevent the assumption of bad positions during the hours of school work and in reading. Compensation by means of the shoe in the event that there is a short leg is obviously of importance, even though the writer does not believe that this factor enters strongly into the causation of scoliosis with frequency.

In postural scoliosis, as in postural kyphosis, the direct attack upon the deformity must be made by means of gymnastic treatment. The gymnastic treatment cannot be treated in detail in this place, but it will be well to say something of the ends to be attained by it. Long ago, attention was called to the fact that the scoliotic patient has not only a distorted spine, but that his sense of position is abnormal. Having become accustomed to his abnormal posture, he feels crooked when his spine has been placed straight. He requires, therefore, to be re-educated as to his sense of correct posture.* This is best done by placing the patient in correct position in front of a mirror and explaining what are the elements of correct posture to be attained. Such a mirror drill should constitute a part of each gymnastic treatment. The patient should furthermore be taught and encouraged to assume the correct position with the aid of a mirror, in his home. Unfortunately, there is no little misconception of the elements of correct posture among many whose duty it is to assist in bringing it about. Not seldom do we see patients being taught to stand with the shoulders thrown back, the scapulæ brought together, as a means of assuming an erect posture. This is erroneous; it is not the position maintained by persons with normal carriage, nor can it be maintained by anyone for a considerable length of time. The patient should be instructed to carry the head erect, the chin in; the chest should be elevated and the abdomen kept from being prominent in front. If this is done, the position of the shoulders requires no special attention; they will be carried back to a sufficient extent. The patient should be taught to bear his weight equally upon both of the lower extremities. Children with asymmetrical posture quite commonly are found standing upon one leg with predominance and habitually. The necessity of sitting and standing correctly at all times is to be impressed upon the patient repeatedly as a part of each treatment. Here, as in all cases of faulty posture, the success of our efforts depends very largely upon the extent to which we can secure interest and intelligent coöperation. Lacking this, the result will be disappointing in spite of the most intensive gymnastic exercises. This is to be borne in mind in considering the prognosis, particularly in the case of children of low mentality and those who have been excessively indulged by their parents. If the child's general training is to begin with the correction of a postural fault, we shall probably fail in both respects.

The second element in the gymnastic treatment lies in the development of flexibility. In a large proportion of these patients there is a diminished flexibility of the spine; this may be true as concerns flexion toward the side of the convexity of the curvature, as regards motion in extension of the spine, or in both respects. It is to be overcome by means of movements and positions accomplished passively by the gymnast in charge, either with his hands or with the aid of apparatus, or it is to be accomplished by means of asymmetrical exercises with the assistance of the gymnast. Of very great value, after the patient has been under treatment for a time, is the assumption of the positions known as "voluntary over-correction." In the case of the usual left total scoliosis, this position would be attained as follows: By advancing the right foot and slightly bending the knee, the patient drops the right side of the pelvis. By placing the right hand upon the occiput, the elbow held as high as possible, the left hand is pushed against the left lower chest so as to move the spine toward the right side. With practice, the patient may be taught to make these two movements synchronously, with force and by count. If the spine has been made quite flexible, a position of quite definite over-correction can thus be achieved. When properly done this is a most useful maneuver and one which may be utilized with profit as a home exercise, to be continued long after active treatment in the gymnasium has been dispensed with.

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* Roth, "Treatment of Lateral Curvature," London (1899).

Just as in the case of postural kyphosis, and just as we shall find it to be the case with structural scoliosis, a most important function of the treatment by gymnastic methods is to furnish the patient with an improvement of muscular control. Since the condition is essentially one of disturbed symmetry, the temptation is great to strive for an intensive cultivation of those muscles whose strength may be presumed to be below normal; by many the weakness of certain muscles, particularly those of the side of the convexity, is looked upon as the fundamental feature of the deformity. It may not be assumed as having been demonstrated, however, that we are able to develop the muscles of the back in a manner so selective and for this reason asymmetrical exercises are to be looked upon as valuable rather in increasing the flexibility of the spine than as a means of developing one set of muscles at the expense of the others. The description of the exercises found in Lovett's book* is much the best to be found in the literature and it is therefore given verbatim.

Lying on the Face. (IV) Trunk Raising. Position.—The patient lies face downward on a table with the spine straight, the hands on the hips, the scapulæ approximated to each other, and the legs just above the ankles, or the legs may be held by the hands of an assistant. (1) The patient inspires and raises the trunk from the table, hyper-extending the spine as far as possible, keeping the head back and the face up, with the elbows still held well back. (2) The patient breathes out and sinks to the original position (Fig. 167).

This is an extension of the spine from its normal position to extreme hyper-extension in which the spinal motion occurs largely below the tenth dorsal vertebra, where hyper-extension anatomically takes place. The weight of the trunk is raised by action of the back extensor muscles which are very generally called into play. It is a general strengthening exercise for these muscles, but in cases with marked increase of the lumbar curve it must not be used to increase this, in

* "Lateral Curvature of the Spine and Round Shoulders" (1916).

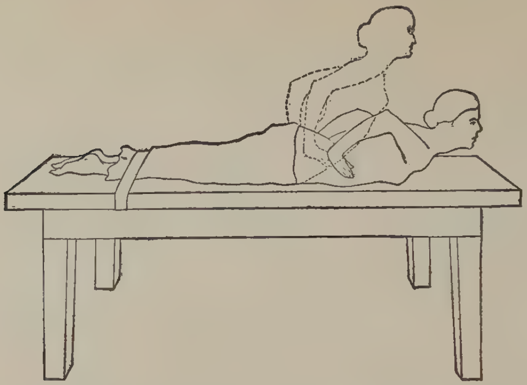

FIG. 167.—Trunk raising. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

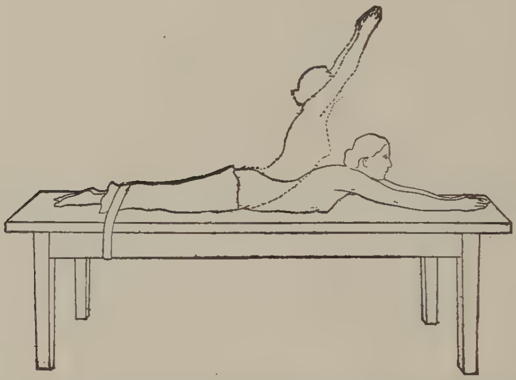

FIG. 168.—Trunk raising. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

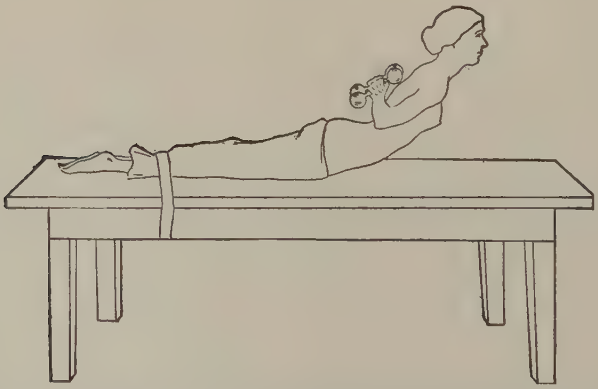

FIG. 169.—Trunk raising. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

such cases Exercise II being available. The latter is probably a weaker exercise, because in it the extensor muscles do not contract to their fullest extent. The exercise may be made harder by placing the hands behind the neck and squaring the elbows back or by extending the arms beside the head, which raises the center of gravity (Fig. 168).

The above may be modified in the following manner: The patient clasps his hands behind his back above the level of the waist-line, with elbows flexed and hand closed against the back, and, as he hyper-extends his trunk, stretches his arms backward forcibly, extending the elbows, and keeping the hands clasped. By this modification the scapulæ and shoulder joints are carried back and the hyper-extension done with an improved position of the shoulders. This is particularly suited to round shoulders.

This exercise may be made stronger by the use of dumb-bells or a staff as indicated in Figs. 169–171.

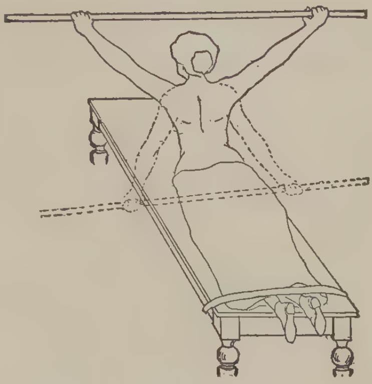

FIG. 170.—Trunk raising. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

Exercises Lying on the Face, the Trunk Projecting Over the End of the Table.—The legs rest on the table, the surgeon making the ankles secure by means of a strap or by holding them. The body above the hip joints hangs over the table end, head downward. The hands are placed behind the neck with the elbows squared back.

V. Trunk Raising from Head Downward Position. (1) The patient inspires, and raises the trunk as far as possible by hyper-extending the hip joints and the spine. (2) During the expiration she sinks to the primary position. The spine should be kept in the mid-plane and the head not allowed to flex (Figs. 172–174).

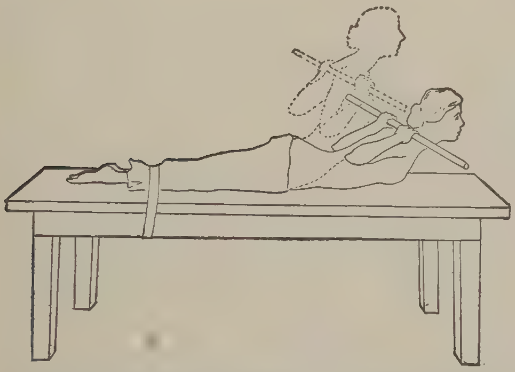

FIG. 171.—Trunk raising. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

This is a spinal extension movement mostly without superincumbent weight, beginning at forward flexion and ending in marked hyper-extension, calling the extensor muscles into activity from a stretched to a completely contracted condition. It thus combines the range of motion in Exercise II with that of Exercise IV. It is a heavier exercise than either. From the start of the exercise till the horizontal position is reached the spinal extensors and glutei are the muscles chiefly active, as the maintenance of balance does not require the contraction of other trunk muscles. The exercise may be made easier by placing the hands on the hips. It is of use as a general strengthening exercise for the back muscles in any case where the patient is strong enough to take it.

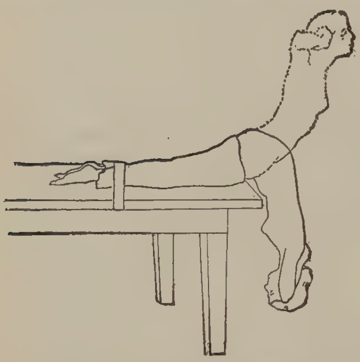

FIG. 172.—Trunk raising from head downward position. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

Exercises Lying on the Back.—The patient lies on a table or on the floor with the head, trunk, and legs straight, and the feet secured either by a strap or by being held. The arms are folded on the chest.

VI. Trunk Raising to Sitting Position.—(1) The patient rises slowly to the sitting position with the spine stiff and not allowed to flex. (2) The patient sinks to the primary position with the spine still stiff, the head touching the table before the back (Fig. 175).

The exercise is made easier by placing the hands on the hips, and harder by placing the hands behind the neck with the elbows squared back. The upright position is brought about by the contraction of the abdominal muscles, which aid in maintaining the upright position, and require exercise in cases of prominent abdomen and of increase of the lumbar physiological curve accompanying scoliosis and round shoulders.

Miscellaneous Symmetrical Exercises. (VII) Weight Carrying on the Head.—A bag filled loosely with sand, weighing from 3 to 15 lb., is placed on the top of the patient's head, and she walks slowly to and fro with the arms preferably clasped behind the neck and the elbows squared back. The exercise may be made more difficult by having the patient walk on tiptoe. The attitude assumed should be as erect as possible and the weight as heavy as can be carried steadily.

It is a matter of common information that the habitual carrying of baskets and loads upon the head induces an erect carriage and a straight spine. The presence of weight upon the head necessitates holding the spine as straight as possible under the weight, as it is thus most economically carried, and this instinctive adjustment to superincumbent weight is depended on for its corrective effect. The exercise is suited to mild cases with noticeable bad carriage and poor balance.

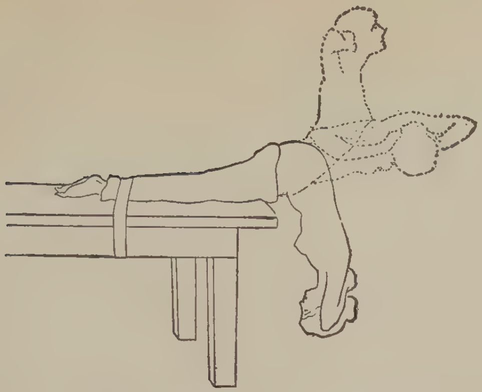

FIG. 173.—Trunk raising from head downward position. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

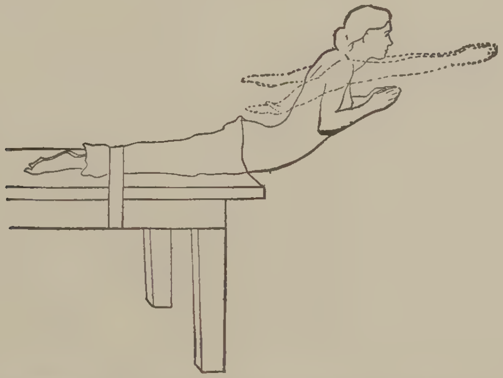

FIG. 174.—Trunk raising. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

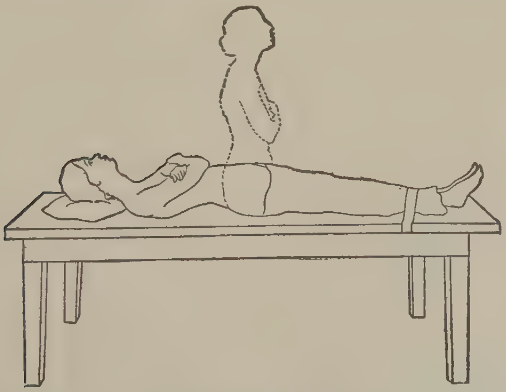

FIG. 175.—Trunk raising to sitting position. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

VIII. Mirror Self-corrective Exercise.—The patient, bared to the hips, faces a mirror in front of which hangs a plumb line. The patient then stands in such a position that the plumb line cuts the middle of the pelvis, and by a muscular effort brings the middle of the thorax and the vertical line of the face as nearly as possible under the plumb line, bringing three important landmarks into the median line of the body, thus securing an improved position. This is held for a few seconds and then the relaxed position resumed. The exercise is repeated several times, the improved position being held longer each time.

The exercise is a muscle training and is not in any way a mobilizing exercise, but enables the patient to associate a certain position with a certain muscular effort, and is of great value in enabling patients to identify by muscular sense the corrected position. The exercise requires but little effort and may be done at home without assistance. It may be modified in various ways by adding free-arm staff, or dumb-bell exercises, which change the center of gravity, strengthen muscles approximating the scapulæ, and prolong the corrected attitude.

Asymmetrical Exercises. IX. Hip Sinking (Hoffa). Position.—From the fundamental standing position the patient advances the foot, on the side opposite to the convexity of the lateral curve, forward and outward about two foot-lengths. (1) The patient bends the forward knee, sinking the hip on that side. (2) The patient resumes the primary position (Fig. 176).

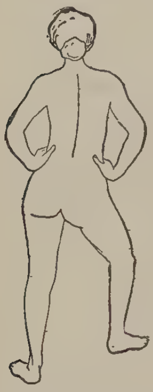

Fig. 176—. Hip sinking. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

A passive side correction of the lumbar curve, due to a lowering of the pelvis on the side of the advanced leg when the knee is bent. Suitable for lumbar curves.

X. Self-correction (Lorenz).—The patient assumes the fundamental standing position and places the hands on the side to which the dorsal spine is convex upon the side of the thorax opposite to the greatest dorsal curve; the other hand is then placed on the ilium. (1) By a side thrust of the hand on the thorax the patient corrects the dorsal curve as much as possible, maintaining the correction for a few seconds. (2) The patient relaxes to the primary position. The exercise may be modified by placing the hand on the side to which the dorsal spine is concave on the top of the head, as it thus tends to raise a low shoulder. The rest of the exercise is performed as described (Fig. 177).

A side thrust of the dorsal spine with pressure applied to the convexity of the dorsal curve against resistance furnished by the other hand on the ilium or the head. Suitable for dorsal scoliosis, but not powerful, and useful as a means of stretching; chiefly good because it can be done by the patient unaided at frequent intervals.

Exercises IX and X may be combined for a double curve with one element dorsal and the other lumbar.

XI. Hip Sinking from Stool. Position.—The patient stands erect on a stool on one foot (the foot on the side of the convexity of the curve).

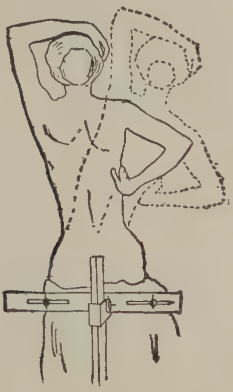

Fig. 177.—Self correction. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

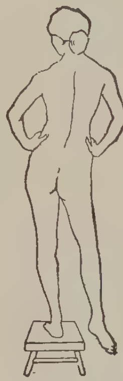

Fig. 178.—Hip sinking from stool. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

(1) The patient lets the free leg sink as much as possible, thus lowering the pelvis and hip on that side. The knee of the supporting leg must be kept straight. (2) The patient resumes the original position (Fig. 178).

A passive side stretching of the lumbar curve suitable for lumbar scoliosis. The leg and pelvis drag down on the side of the concavity of the lateral curve, tending to stretch contracted structures and straighten the curve.

XII. Trunk Hyper-extension with Side Bending.—Lying on the Face. The patient lies face downward on a table or on the floor as described in Exercise VII. (1) The trunk is raised from the table as far as possible by hyper-extending the spine. (2) From this position the trunk is bent to the side toward which the lumbar curve is convex. (3) Position 1 is resumed. (4) The prone lying position is resumed (Fig. 179).

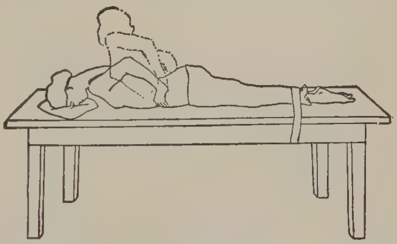

Fig. 179.—Trunk hyperextension with side bending—lying on face. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co,. Phila.)

This exercise is an active lateral flexion of the spine in the position of hyper-extension. As hyper-extension locks the dorsal region against side flexion, the movement is almost wholly confined to the lumbar region. If there is a right dorsal curve in connection with a left lumbar curve, bending to the left, while it corrects the lumbar curve, does not at the same time greatly increase the dorsal curve, as that part of the spine is locked against side bending. The exercise is, therefore, suited not only to lumbar curves, but especially to compound curves in both dorsal and lumbar regions.

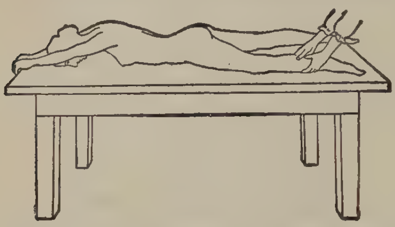

Fig. 180.—Drawing up the hip—lying on the face. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

XIII. Drawing up the Hip. Lying on the Face. Position.—The patient lies prone on a table, holding the end with both hands, the arms extended and the spine and legs in a straight line. (1) The surgeon grasps the ankle on the side of the lumbar convexity and resists while the patient draws the hip up as far as she is able, the knee being kept straight. (2) Position 1 is resumed (Fig. 180).

The approximation of the side of the pelvis and the thorax on the side to which the lumbar curve is convex is brought about by an active contraction of the muscles on the convex side of the lumbar curve which it is desirable to develop. The amount of work thrown on these is determined by the amount of traction made on the ankle. The exercise is suited to cases of lumbar curves or to the lumbar element of compound dorsal and lumbar curves.

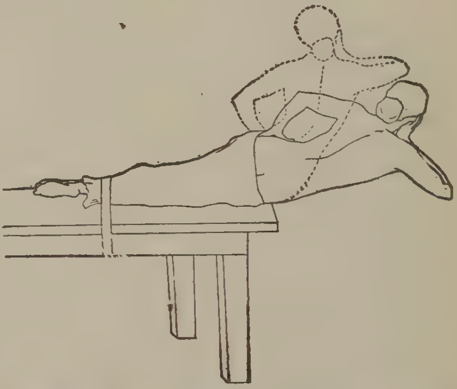

Fig. 181.—Side flexion of the trunk from the side-lying position. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

XIV. Side Flexion of the Trunk from the Side-lying Position. Position.—The patient lies on a table with the concavity of the lateral curve downward and the trunk projecting over the edge of the table above the pelvis, the patient being supported in this position, and the ankles secured by means of a strap. The spine is held in medium extension, the upper hand on the hip and the lower hand on the back of the neck. (1) The trunk is bent laterally and upward as far as possible. (2) The original supported position is resumed (Fig. 181)

In this exercise the weight of the trunk is thrown on the muscles of the convex side of the lateral curve. The raising of the trunk tends both to diminish a curve existing near the dorsolumbar junction and to exercise strongly the muscles which aid in its correction. It is suited to total, lower dorsal and dorsolumbar curves.

XV. Trunk Bending to Both Sides with Hand Pressure (Mikulicz). Position.—In the case of a right dorsal left lumbar curve the patient places the right hand on the prominence of the ribs just under the shoulder blade, and the left above the ilium on the lumbar curvature. (1) She then bends the body slowly to the right side, while the right hand and thumb press against the dorsal prominence. (2) The upright position is resumed. (3) The patient bends to the left and backward, pressing with the left hand against the lumbar curve. (4) The upright position is resumed (Fig. 182).

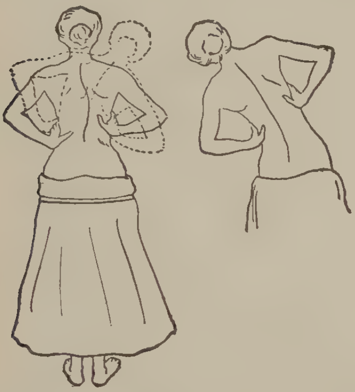

Fig. 182.—Trunk bending to both sides with hand pressure. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

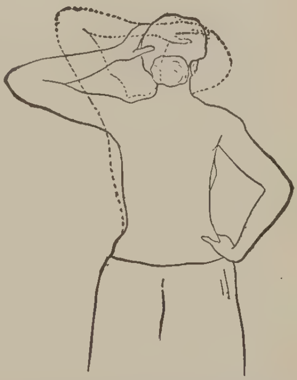

Fig. 183.—Passive head side bending. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

This is a combined mild active and passive correction for a double curve. Opposing forces are applied to the convexities of the curves, thus tending to straighten the spine, which is at the same time bent by means of muscular action, first to the side of the convexity of the dorsal curve and then to the side of the convexity of the lumbar curve.

XVI. Passive Head Side Bending. Position.—The patient stands with the hand on the side of the concavity of the lateral curve against the side of the head above the ear. (1) The head is pushed as far as possible to the side that corrects the curve. (2) The original position is resumed (Fig. 183).

A passive correction of the cervical lateral curve by a side bend of the upper part of the cervical region which tends to diminish the curve. Of use in cervical and cervicodorsal curves, either alone or existing in combination with others.

XVII. Trunk Raising with Asymmetrical Position of Staff—from Prone Lying Position. Position.—The one described for exercises with the patient lying on the face (Exercise VII with a staff grasped in both hands, the arms being extended beside the head). (1) The trunk is raised from the table and the staff brought over behind the head obliquely, the hand on the side of the convexity of the curve being carried down toward the feet, and the other carried up over the head until the staff is brought as nearly as possible into the long axis of the body and pressed against the back. (2) By a reversal of the movement the original position is resumed (Fig. 184).

The scapula on one side is raised, and the position of the staff tends to correct an existing curve in the dorsal region. The exercise amounts to a spinal hyper-extension in a corrected position of the dorsal spine.

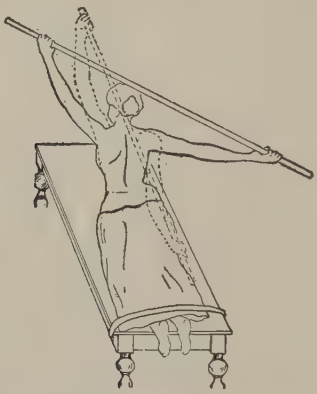

Fig. 184.—Trunk raising with asymmetrical position of staff—from prone lying position. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

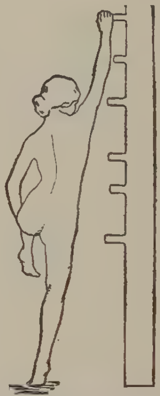

Fig. 185.—Partial suspension by one arm with other arm and leg locked. (From Lovett's Lateral Curvature of the Spine and Round Shoulders, Blakiston's Son and Co., Phila.)

The exercise is suited to total curves, to simple dorsal curves, and to compound dorsal and lumbar curves.

XVIII. Partial Suspension by One Arm with Other Arm and Leg Locked. Position.—The patient standing by a ladder or under a bar that can be reached without rising on the toes, grasps one rung of the ladder or the bar with the hand of the side to which the spine is concave. On the opposite side, the convex, the arm passes under the knee, the thigh being flexed at the hip, and the shoulder and pelvis are thus approximated. (1) The patient thus standing on one leg flexes that knee and allows the body-weight to come upon the arm. (2) The original position is resumed (Fig. 185).

When the arm is placed under the knee the pelvis and shoulder are approximated on that side and the spine made convex to the other side as far as it will go. The structures on the concave side are thus put on the stretch and, by allowing the body-weight to come on the arm holding to the ladder, a further stretching force is exerted on the structures on the concave side. The exercise is suited to total and dorsal curves.

Structural Scoliosis.—From what has already been said in connection with postural or functional scoliosis, it will be observed that whereas spinal asymmetry or curvature of marked degree may exist for years without presenting the development of recognizable structural change in the bones of the spine and chest, there are many other cases in which such structural change is apparent very early; that is to say, such changes are already present when the deformity is first noticed by the parent and physician and very often indeed at a time when the deformity is still of slight degree in all of its elements. As the result of careful observation and graphic record of many cases, it must be accepted as true that there is no definite relationship between the degree of general asymmetry of the body and the extent of bony distortion; it will be necessary to emphasize again, later in this text, that there is no definite and constant relationship between the individual elements of bony change from case to case or as concerns the involvement of different segments of the spinal column. These are, in the mind of the writer, weighty reasons for considering structural scoliosis as an entity of distinctive character; perhaps it were better to say that this is the justification for giving postural scoliosis separate consideration.

It has already been stated that the characteristic which distinguishes postural from structural scoliosis is that the structural form exhibits definite alterations in the bones of the spine and the chest which are not materially modified by removing the superincumbent weight from the spine. Because the sideward bend, or lateral deviation, is always accompanied by a twist or torsion element this form of scoliosis has also been called "rotary lateral curvature." The torsion, also spoken of as rotation, deformity involves the bodies of the vertebræ and carries with it the ribs; there is thus produced a characteristic deformity of the chest wall which is observed to best advantage either in the forward bending position of the body, or with the body lying prone on a hard plane surface. The vertebral bodies are always twisted towards the side of the convexity of the lateral curve of the spine. This is most often toward the right in the dorsal segment and toward the left in the lumbar. As regards the degree of fixed or structural deformity, there is the greatest variation to be observed; this concerns not only the amount of general deformity, however, but also the relationship between the elements of lateral bend and torsion. At the same time these two elements are always seen in combination in structural scoliosis. The twist of the line of vertebral bodies on its vertical axis constitutes, therefore, the most distinctive feature of structural scoliosis. The physiological anteroposterior curves may be but little changed, they may be much exaggerated or they may be considerably diminished in degree. Thus, the backward curve in the dorsal region may be intensified; in this case the term "kyphoscoliosis" is often used. Occasionally the dorsal segment is much less kyphotic than normal, the so-called scoliotic flat back.

In dorsal scoliosis there is always marked distortion of the thorax; a distortion which stands in a fairly definite relationship to the degree of torsion deformity in the spine itself. The vertebral body, twisting toward the convexity, may be said to carry with it the rib whose angle moves backward on this side. There results a marked lengthening of the one horizontal diagonal and a shortening of the opposite diagonal in accordance therewith. This is best illustrated by the well known schematic drawing of Lorenz which shows how the chest capacity is diminished on the side of the convexity, the projection backward of the line of rib angles on this same side, constituting the so-called rib hump; this also shows the prominence anteriorly of the ribs of the side of the concavity. Lovett has aptly said that "the thorax as a whole tends to retain its normal position with regard to the frontal plane of the body more closely than does the spine, which as it were, rotates in the thorax."

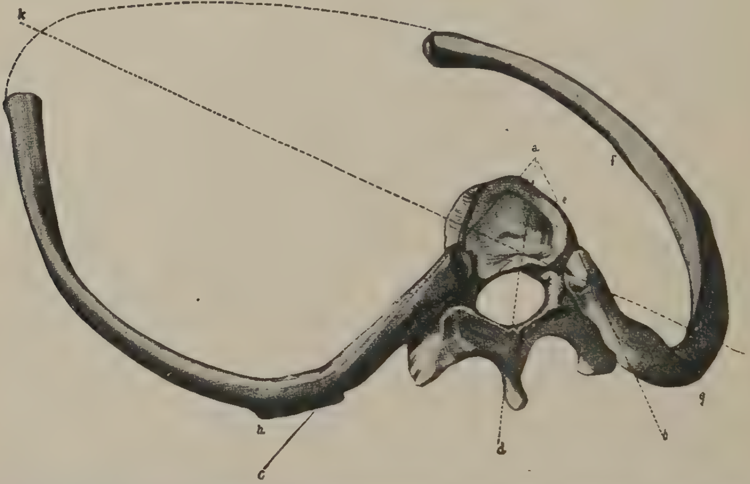

FIG. 186.—Lorenz' diagram of the scoliotic "rib ring," showing how the one diagonal of the chest is lengthened and the other shortened. (From Joachimsthal, Handbuch d. Orthop. Chir. I, part 2, p. 672.)

The individual vertebræ are deformed in their various parts in accordance with the degree of the deformity of the spine. The bodies assume the shape of a wedge at the apex of the convexity, a wedge in which the torsion element is, however, also expressed. Just as the transverse processes move backward carrying with them the angles of the ribs, in the dorsal segment, so in the lumbar region do they move backward on the side of the convexity and, therefore, most often towards the left. This becomes manifest in the appearance of a rotation hump in the loin. The spinous processes, because of the ease with which they may be localized by the finger, are commonly used as the means of measuring and recording the lateral deviation. While a more practical method for doing this cannot be proposed for the present, it must not be overlooked that there is a deflection of the spinous processes toward the convexity as a rule; a deflection which is not of constant degree, however, and this fact serves to impair the value of the line of spinous processes as the measure of the lateral deviation.

As may be expected, the soft parts connected with the spine exhibit marked abnormalities. This is especially true and important in respect of the muscles of the back. Many years ago muscular contractures on the side of the concavity were considered by some to be the basic factor in the production of scoliosis and myotomies were done in consequence and to no purpose. A very exhaustive study of the musculature in scoliosis is that of Schulthess.* He has been able to show that the condition of the muscles, instead of being causative by reason of their abnormal physical condition, is rather expressive of the state of their function as influenced by the deformity which has developed. Where this has interfered with muscle function atrophy and degeneration have followed, by reason of long continued inactivity, regardless of whether the muscles of the convexity or the concavity are concerned. In the muscles in the "dead angle" of the concave side where there is practically no motion owing to what really amounts to ankylosis of the spinal joints, there is found complete fatty degeneration with slender streaks of tendon tissue. On the convex side the muscles are characterized by extensive fibrotic changes; these degenerations are to be expected only in cases of severe deformity in which marked interference with movement of the spine has resulted. On the other hand, in cases less severe and with less impairment of motion, there is often observed hypertrophy of muscle on the convex side; this may be ascribed to the functional response to the drag made upon the muscles in consequence of the overhang toward the concave side. It is only to be expected that contractures of muscle will be found at times on the concave side, which may act as hindrances to correction of the deformity; these are to be regarded as the consequence of functional inactivity, of the fact that the muscles have not been called upon to stretch and not as a cause of the deformity in the first place. Another careful anatomical study of the musculature of scoliotics has been published by Hans Virchow.† His conclusions are in substantial agreement with those of Schulthess; the changes in the muscles are to be regarded as the consequences of the spinal deformity and not as a cause thereof. He makes the interesting and important additional statement, however, that our knowledge of the functions of the deep muscles of the back is as yet too meager to make it easy to interpret the significance of the changes found in them, in practical terms.

* *Cf.* Handb. d. Orthop. Chir. Joachimsthal, ii (Jena, 1907).

† Zeitschr. f. Orth. Chir., Bd. xxxiv (1914).

In scoliosis of long standing and of marked degree, changes of other parts of the skeleton are commonly seen; if the upper segment of the spine is involved, asymmetry of the calvarium and the face may occur; if the lumbar segment is concerned marked changes of the pelvic bones may take place producing the oblique or scoliotic pelvis spoken of by obstetricians. In dorsal curves with marked torsion deformity, the scapula assumes abnormal positions; on the convex side it swings away from the vertebral column and on the concave side it approaches the spine. Its axis may move around so that it may even lie horizontally.

In consideration of the very great distortion of the trunk which is often seen in severe structural scoliosis, it is not surprising to learn that important displacements of internal organs occur with consequent functional disturbances and organic damage. The article of Bachmann* is the only investigation embracing a large series of post-mortem examinations to be found in the literature. Displacement of the heart toward the concavity, dilatation of the right side of the heart, interference with the normal position of the aorta, esophagus and the lungs, are described by him. He concludes that severer grades of structural scoliosis must be regarded as a distinct menace to the normal expectancy of life.

The Etiology and Nature of Structural Scoliosis.—In discussing the essential character of structural scoliosis it is first of all requisite to realize that however much resemblance there may be between the various cases when looked upon in a superficial manner, identical external appearances may be presented by spines deformed as the result of processes entirely different in their nature.

Congenital Scoliosis.—Congenital scoliosis is by no means unusual, being recognizable as a deformity at birth, soon afterward or only after months or years, depending upon the degree of deformity as well as the association with other congenital anomalies. Concerning the causes of such deformities we are just as wise as in the case of other congenital deformities and no more so. Persistent and abnormal position of the fetus in utero may be regarded as the probable cause of some of the simpler ones; for the explanation of the cases complicated by associated deformities we must enter the realm of speculation. Most often the *x*-ray will show the existence of malformed vertebral bodies, of supernumerary ribs either cervical or lumbar, of fusions or rudimentary formations of ribs. The combination of congential scoliosis with congenital malposition or elevation of the scapula has been observed quite often† (Fig. 187). Cases have also been described in which there was association with anomalous formations of the overlying soft parts. The writer has observed one instance of very severe left dorsoconvex scoliosis, in a young man, in which a very large, soft, fibro-vascular nevus hung down from the apex of the convexity

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* Abt, D. I., "Bibliotheka Medica," Heft iv (1899).

† Freiberg, Annals of Surgery (May, 1899).

(Fig. 188). The nevus was quite large at birth and was the means of attracting attention to the scoliosis at this time.

Acquired Structural Scoliosis.—While the occurrence of anatomical variations of the spinal column has long been recognized as not unusual, Boehm, in 1906, published a study of 20 cases of scoliosis investigated by means of the x-ray in which he found such variations either in the number or formation of the vertebræ and their adnexa. He felt justified, on the basis of this, in considering the so-called habitual scoliosis of congenital origin to this extent.* Boehm's observations have had the effect of attracting attention to the frequent occurrence of such congenital anomalies, as they may be seen during life in x-ray plates. There are weighty reasons, however, for objecting to wide generalization upon this basis. Many cases of adolescent scoliosis are seen in which it is impossible to recognize either numerical variations or asymmetries of undoubtedly congenital nature; even when present, some further explanation is due for the appearance of clinically recognizable deformity of the spine only so late as the second decade of life, since this is the time when the overwhelming majority are so recognized. Furthermore, variations both as regards number and symmetry are not infrequently seen either in the absence of definite

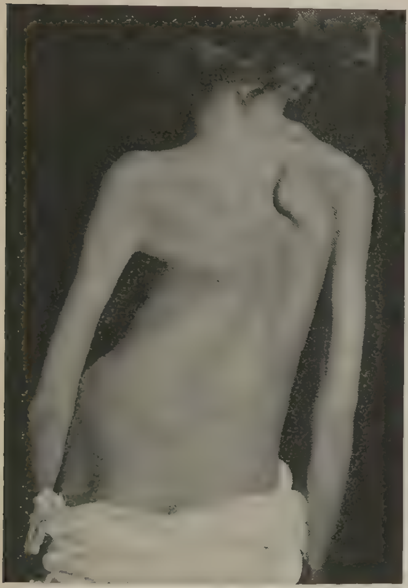

FIG. 187.—Congenital scoliosis with elevation of the right scapula. Girl 12 years old. The x-ray shows both defects and fusion of ribs.

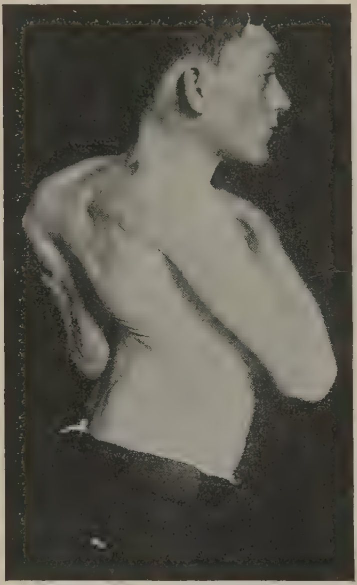

FIG. 188.—Congenital scoliosis in a young man. A large fibrovascular nevus hangs from the apex of the torsion hump on the back.

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* Boehm, Boston Med. and Surg. J. (1906) also Fortschr, a. d. Geb. d. Roentgenstr., Bd. xi.

scoliosis* or in connection with slight curvatures which are obviously lacking the progressive character of adolescent scoliosis. Even though we might be willing to ascribe an important attribute to congenital abnormalities of this kind in determining the *direction* and *degree* of spinal deviations in the adolescent epoch of life, it would seem that there must be some factor responsible for the progressive character of the deformity; a deformity which within a few months may produce quite surprising distortion of the spine and thorax. For Julius Wolff it was enough to assume a high grade of muscular insufficiency;† the element of congenital asymmetry, then not yet under discussion, would have added plausibility to his explanation. At the same time, even to one as sympathetically inclined toward Wolff's theory of functional pathogenesis as the writer, it seems inevitable to conclude that some condition must exist of impairment of bone texture akin to that of rickets in which disease we find strikingly similar structural scolioses both as concerns the character and grade of deformity. In addition, reference must be made again to the studies of scoliotic musculature, which seem to show that the marked changes are to be given the rôle of results rather than of causes of the deformity. Adolescent scoliosis is a deformity which has interesting congeners in the genu valgum, genu varum and pes valgus of adolescence. Peckham‡ believes that the explanation lies in a disturbance of the endocrine balance which takes place at this time. Since it is believed that the whole complex of changes which we know as pubertal consist of phenomena to be ascribed to the endocrine organs, this view has much which seems plausible; at the same time, all of these phenomena are too little understood to form the basis of more than a speculative hypothesis, however attractive. It is nevertheless significant to the writer that most of the cases of adolescent scoliosis which he has observed in late years in private practice, have had their beginnings just prior to or coincident with the first visible signs of impending sexual development.

Acquired structural scoliosis is commonly attributed to other causes which would seem to play a similar rôle to the congenital asymmetries, as above described; such are distortions of the pelvis, torticollis, unequal vision or hearing. Rickets has already been referred to as a frequent cause of marked scoliotic deformities in early life; numerous authors regard the adolescent form as nothing but recrudescence of pre-existent rickets. Osteomalacia has long been known as the cause of severe scoliotic deformity; it is not usually seen before the adult period of life. Empyema is a not infrequent cause of scoliosis; the contraction of the fibrous or cicatricial masses in the pleura pulls the spine toward the affected side, so that we may have dextroconvex curve with left empyema and *vice versa* (Fig. 189).

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* Adams, Bost. Med. Surg. J., clxii (1910) p. 569.

† Freiberg, editorial article, Annals of Surgery (July, 1897).

‡ Journ. A. M. A. (Oct. 13, 1917).

Infantile paralysis or anterior poliomyelitis is a frequent cause of structural scoliosis, often of the most severe kind. Aside from acting as a contributory cause by producing shortening of one lower extremity or necessitating a faulty attitude by paralysis of an arm, it may result in paralysis of the muscles on one side of the spine. Lovett* says that "it does not follow that paralysis of the muscles of one side of the back is followed by a curve convex toward the paralyzed muscles, as would naturally be expected. The curve is the result of the effort of the patient to adjust his center of gravity to the new conditions induced by unilateral paralysis. This equilibration may result in a curve convex to the right or left in a right-sided paralysis." Scoliosis may also be found as the accompaniment of other nervous disease, such as spastic paralysis or other paralyses, whether transitory or permanent. The view that most cases of adolescent scoliosis are the result of hitherto unrecognized poliomyelitis, which has been advanced by Hibbs, does not seem tenable in the light of what knowledge we have. We must also agree with Lovett in discrediting habit or occupation alone as the cause for obviously structural scoliosis. Some other factor is required to explain the structural changes which are found; we should otherwise find it still more common than is the case in view of the great frequency with which occupations require the assumption for long periods of scoliotic postures.

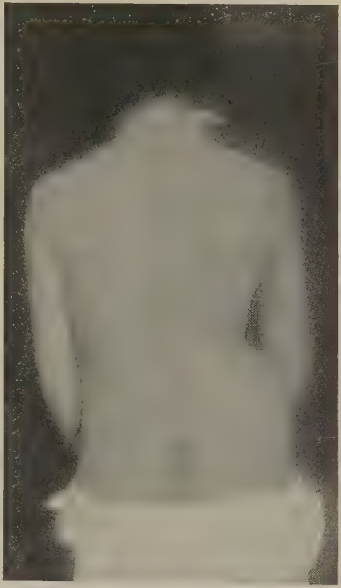

FIG. 189.—Left dorsolumbar scoliosis, following empyema of the right side. Girl of 12 years. The operation scar is visible.

Examination and Methods of Record.—What has been said of the etiology of scoliosis will make it apparent that inquiry into the personal history of the patient is desirable in order to assist in establishing the nature of the process which has given rise to the deformity. The existence of rickets in infancy and early childhood should be inquired into, both by ascertaining such signs as late teething, bowel disturbances, sweating of the head, as well as by seeking for the vestiges of the disease in bony deformities such as bow legs, rachitic rosary and enlarged epiphyses of the long bones. It is very important to determine the character of acute illness which may represent a causal factor, such as poliomyelitis or empyema, and as well to examine carefully for the residual deformities of such. Careful note should be made of the general condition of the patient, both as regards musculature and carriage of the body. The heart and lungs should be carefully examined. Asymmetry of the lower extremities is important to recognize and careful measurement is called for, with this object in view.

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* Lateral curvature, *loc. cit.*

It is always difficult to ascertain the length of time which has elaspsed since the deformity first became apparent; it is common to see children with quite high degrees of bony change, whose mothers assert that these have positively taken place within an astonishingly short period. One can scarcely credit the possibility of the development of such grades of bony distortion within a few months' time. Nevertheless in some cases it seems inevitable to accept the statements of very intelligent and careful mothers, to this effect. The degree to which the secondary sexual characteristics, such as the growth of axillary and pubic hair and the development of the mammæ, have progressed is of some importance and especially with regard to the prognosis. It would appear that the nearer the child is to complete sexual maturity, the less is the likelihood of further progress in the deforming process; on the other hand, the farther we are away from complete maturity the greater the need for energetic corrective measures and the greater the chance for therapeutic success.

The Examination of the Spine.—It is not necessary to subject the patient to the feeling of uncomfortable exposure in order to make a satisfactory examination. At the same time, the spine should be completely exposed from the neck to the cleft of the nates. An apron or the child's own garment may be hung from the neck so as to cover the front of the trunk; the clothes should be fastened by means of a strap around the hips so that they are secure from slipping down. The shoes should be removed and the patient be made to stand with the feet close together, the arms hanging easily at the sides; it is desirable that the light from a window should fall squarely upon the back so as to cast no shadows. The patient should be permitted to stand in this position quietly for a few minutes until the natural and unconstrained posture has been assumed. Note is then made of the contours of the trunk from axillæ to hips. In a normal child the waist angle of the two sides should be equal; inequality in this respect indicates an inclination or displacement of the trunk toward the side of the more acute angle. At this time we may also note the line joining the tips of the shoulders, the position of the scapulæ and of the head; by stepping to the side the anteroposterior curves of the spine may be observed.

Estimation and Record of the Elements of Deformity.—The progress of a patient with structural scoliosis, for better or for worse, must be measured by the lapse of years; it should most often embrace the period beginning with the onset of adolescence to the cessation of growth. The child has during this time become an adult. It is therefore obviously desirable to have the elements of the deformity recorded in such a manner that comparison of the condition may be made with ease and a fair degree of accuracy, as it has been recorded at various times during this long period. The writer would also emphasize the desirability of possessing a graphic record from the time of the first examination, whether the patient is submitted to treatment immediately, or not. In his earlier experience he has had abundant opportunity to regret neglect of this precaution; there was thus lost the opportunity of demonstrating to himself and others what had really taken place during a long interval, either in the absence of treatment or as the result of methods to be either approved or condemned, but satisfactorily so only on the basis of a really trustworthy graphic method of record. It is certain that this can not be done by means of the unaided memory or simple measurements. At the same time, it must be said that the measurement and record of scoliosis presents a problem which no one has thus far succeeded in making at once simple and accurate; this is attested by the fact that nearly every one who has given serious consideration to the matter has evolved his own method. As may be imagined, the objections to the various methods lie either in the complicated character of the apparatus required, the difficulties involved in their use, or on the other hand in a degree of inaccuracy which would deprive them of the title to usefulness. It will have to be confessed that much elaboration in the interest of meticulous accuracy cannot be justified; to accomplish such accuracy upon the dried skeleton would be far from easy and in the living subject it is simply not possible within the limits set by the conditions under which patients have to be examined and the variations in the skill and patience of the examiners. The record by photography has been the easiest response to these difficulties in the opinion of most men; either in the form of simple views of the unclothed figure, posed according to standards or not, or by methods involving the use of accessory apparatus. There can be no question but that the photograph constitutes what is the most satisfying kind of record for most persons and especially so for laymen. When made according to a standardized method, like that of Osgood and Bucholz* it may be very accurate, but there are certain disadvantages inherent to it which cannot be brought below this minimum; the production of a negative makes it impossible to avail one's self of the record within a reasonable length of time and a considerable technical equipment is required if the work is to be done under one roof; the result is a picture, which however standardized and however attractive to the eye, is scarcely to be compared, as a measure of deformity or as the token of what has occurred after the lapse of time, with a record capable of being expressed in figures, with or without the production of a graph. By some, the *x*-ray plate is looked upon as the most reliable and most accurate form of record in scoliosis. It gives what is unquestionably the most striking representation of the structural changes which have taken place; it may be taken in the standing position and thus show what is the effect of weight-bearing upon the spine. At the same time it has the same disadvantages as the photograph, in that it gives us no values which are easily compared and that it requires the preparation of a developed plate. At the same time it has been of inestimable value in showing us the error of believing that we had secured over-correction of the elements of deformity, insofar as we are able to produce a picture of the spine through the plaster jacket; something which can be done in no other manner. At the present time this is the only purpose for which the writer employs the *x*-ray in structural scoliosis. During the five years of life, most important to consider in this connection, between the ages of 10 and 15, the trunk grows usually not less than 10 per cent. of its length; the deviations of the spine as seen in the same individuals at the beginning and the end of this period are then subject to at least this amount of variation. For this reason, it would appear that we should have as the measure of the elements of deformity, not simply curve delineations or photographs of such curves, but rather data of comparable values; amount of torsion should be expressed in degrees of the circle, as was long ago recommended and has been made practical by Schulthess by means of his "Nivelliertrapez" (Fig. 190). Lateral deviation of the spine, on the other hand, can be expressed in similarly comparable terms by reckoning the deviation of the line of spinous processes to one side or other of the ideal perpendicular on some such basis as that of percentage. In such reckoning 100 would represent the length of the "axial line;" the line joining the spinous process of the first dorsal vertebra (for practical purposes) and the upper extremity of the cleft of the nates. For

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* Am. Journ. Orth. Surg. (1914).

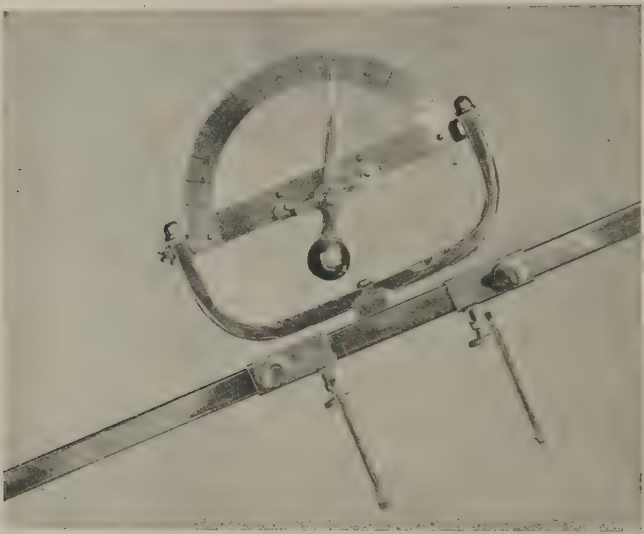


FIG. 190.—The Orthopaedic Theodolite, for measuring variations from the horizontal in any part of the trunk. (Freiberg's model.)

example: In a spine whose axial line measures 50 cm., a deviation of 2.0 cm. will be spoken of as 4 per cent. and if its apex be found at 13 cm. from the top and to the right it may be recorded thus: Deviation 4 per cent. at 26 per cent. to the right. The lateral deviation is always reckoned from a perpendicular erected from the cleft of the nates (Fig. 191). By such a method of record we may not only compare the deviation of the same spine at different times of the patient's life, but we at the same time have established a means of comparison for different spines; when we say 4 per cent. at 26 per cent. to the right, this expresses the degree of lateral deviation quite as well for the spine of a five year old as for a 15 year old child. There remains now to describe how this plan of record may be utilized in practice.

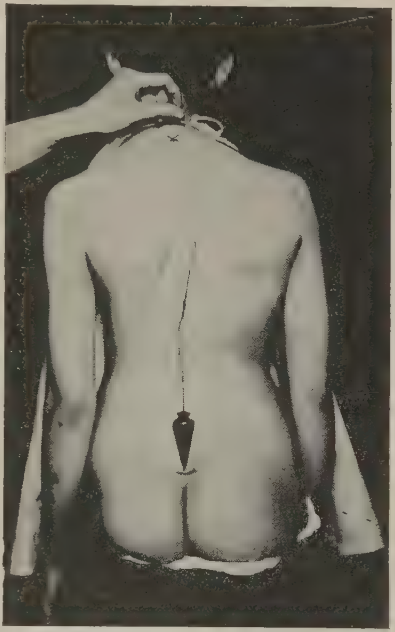


Fig. 191.—Scoliosis measurement and record. Deviation from the perpendicular.

The method to be described has been used by the author for over 10 years quite constantly; very often it has been supplemented by the use of x-rays and photographs of the figure, since both are constantly available to him. At the same time and perhaps because it is the child of his own mind, the method has appeared to be much more satisfactory as a means of orientation and comparison than either of these. The materials required are very inexpensive and easily obtained and the record is available in a few minutes time without having to wait for a process of development. The record may be preserved for future comparison in the form of a graph or not, as may be desired.

For the record of lateral deviation, the following materials are required: A pencil for writing on glass (used on the skin also), a plumb line and bob (Fig. 191), a sheet of transparent celluloid such as used for automobile lights, 60 by 15 cm., with a black line scratched down the middle of its length and provided with tapes at one end for fastening it about the neck; a panel board, 60 by 15 cm., to which is glued a sheet of cross-section paper ruled in centimeters and millimeters. A black line should be ruled down the middle to correspond with the line on the celluloid sheet (Fig. 192). Clamps to hold the celluloid sheet in contact with the cross-section paper so that the black line on each, representing the perpendicular, is in register. It is most convenient to have measurement blanks for record similar to that shown in Fig. 193. A good slide rule should be at hand for bringing all measurements to scale of 50 cm. (or 100 cm. if preferred). If graphic records are desired, these are best made on sheets of translucent cross-section paper cut 60 by 15 cm. They may then be compared by holding them against the light, superimposed. All of these materials may be purchased from dealers in draughting supplies, with the exception of the panel and the measurement blanks, which must be made to order.

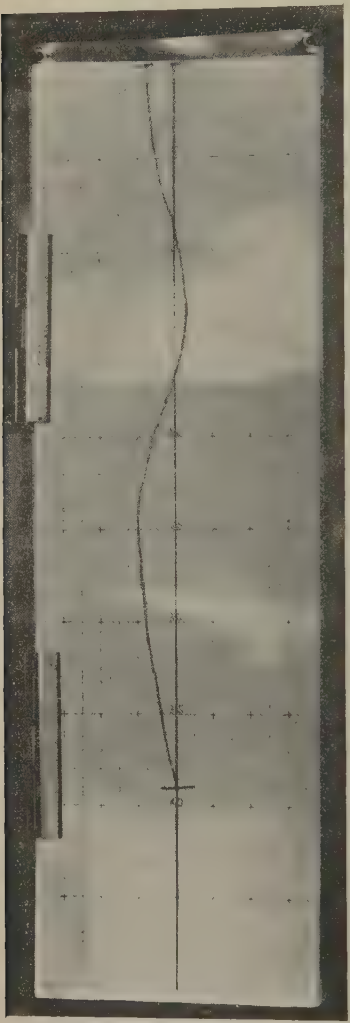
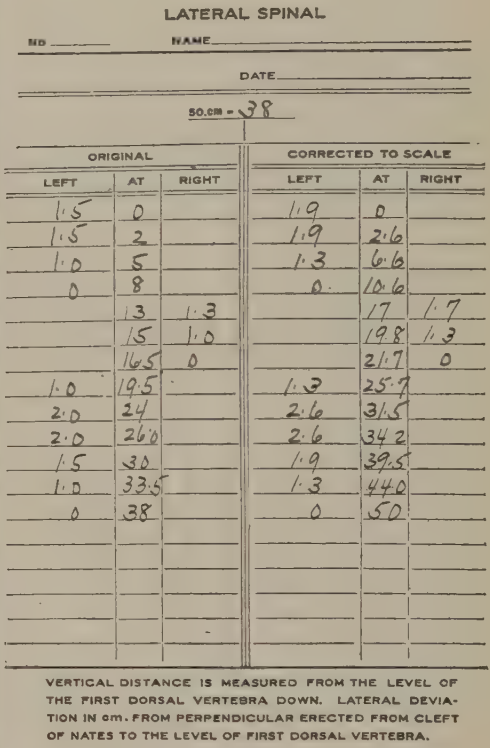


FIG. 192.



LATERAL SPINAL

NO ______ NAME ______

DATE ______

50.cm = 38

ORIGINAL			CORRECTED TO SCALE		
LEFT	AT	RIGHT	LEFT	AT	RIGHT
1·5	0		1·9	0	
1·5	2		1·9	2·6	
1·0	5		1·3	6·6	
0	8		0·	10·6	
	13	1·3		17	1·7
	15	1·0		19·8	1·3
	16·5	0		21·7	0
1·0	19·5		1·3	25·7	
2·0	24		2·6	31·5	
2·0	26·0		2·6	34 2	
1·5	30		1·9	39·5	
1·0	33·5		1·3	44·0	
0	38		0	50	

VERTICAL DISTANCE IS MEASURED FROM THE LEVEL OF THE FIRST DORSAL VERTEBRA DOWN. LATERAL DEVIATION IN cm. FROM PERPENDICULAR ERECTED FROM CLEFT OF NATES TO THE LEVEL OF FIRST DORSAL VERTEBRA.

FIG. 193.

FIGS. 192, 193.—Scoliosis measurement and record. The celluloid sheet with the tracing of the lateral deviation, clamped to the panel board. The measurement card, showing this same curve both as original and brought to scale.

The measurement and record are proceeded with as follows. With the glass writing pencil the spinous processes are carefully marked, from the first dorsal down and a cross mark is made at the upper extremity of the gluteal fold. The plumb line is now held so that it cuts the gluteal fold below at the level of the mark which has been made here; a mark is made on the skin at the level of the first dorsal vertebra where the plumb line indicates. The celluloid sheet is now tied about the neck in such a way that the black line on it coincides exactly with the mark indicating the line of the perpendicular at the level of the first dorsal spine. When there is much deviation from the perpendicular this will naturally cause the celluloid to be quite asymmetrical in position. The lower end of the celluloid is now held so that the black line cuts through the gluteal fold at its upper extremity and with the glass pencil a horizontal mark is made on the celluloid, indicating the lower end of the curve. Carefully sighting with the eye always on the level of each spinous process, the curve is now marked on the celluloid from the first dorsal downward. We now have the curve recorded on the celluloid in its relation to the perpendicular (Fig. 192). The celluloid is now superimposed upon the panel so that the perpendicular lines are exactly in register and it is clamped in this position. We may now read and record in centimeters and millimeters the deviations to the right and to the left of the perpendicular at various points from the first dorsal downward and record them on the left side of the

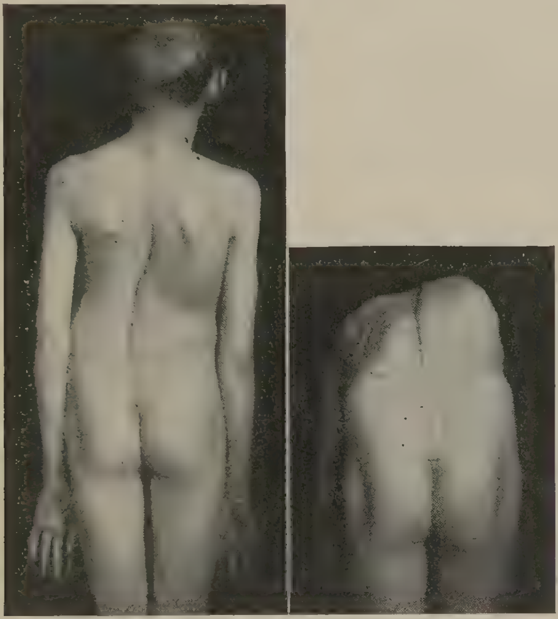


Fig. 194. Fig. 195.

Figs. 194, 195.—Structural scoliosis of rachitic origin in a boy of 10 years. The lateral deviation to the right, in this case, is 4.4 per cent. at 36 per cent. The torsion deformity, shown in forward bending, represents 19°.

measurement card (Fig. 193). By means of the slide rule these measurements are now brought to scale. Only one setting is required and the different values as read from the rule are now recorded on the right side of the measurement card. The use of the slide rule is best learned by personal explanation or from the manual which is furnished with it by the dealer. As used for this purpose, it is quite easy to learn. In order to make the graph, a sheet of translucent cross-section paper is clamped in register on the panel and from the measurement card each measurement on the right side (as corrected to scale) is marked in its proper position by a dot; when the dots are connected by a curve we have the record of the lateral deviation drawn to scale in relation to the perpendicular and it may be filed with the measurement card for future reference.

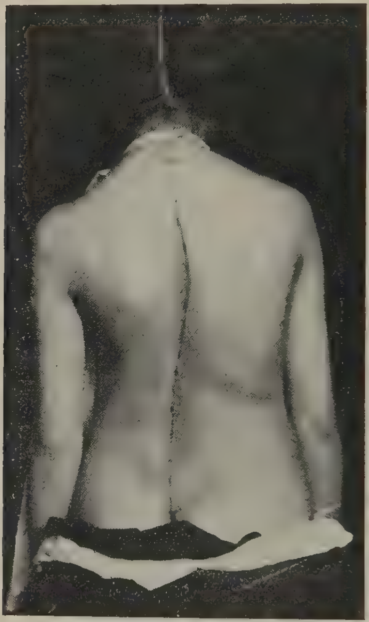


FIG. 196.—Structural scoliosis of the adolescent type; girl of 15 years. The lateral deviation, in this case, measures 6.8 per cent. at 31.2 per cent., to the right.

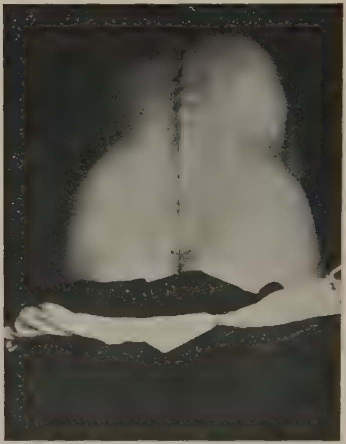


FIG. 197.—Structural scoliosis of the adolescent type. The same patient as in Fig. 196, in forward bending position. The torsion deformity measures 16°, to the right.

In case a number of measurements must be made on the same day, it is convenient and very inexpensive to have a number of the celluloid sheets on hand; marked with the name or number of the patient, they may be left for bringing to scale at any convenient time. It should be added that the record made by the glass writing pencil may be erased by simply wiping off with a tuft of dry cotton.

As mentioned above, the writer considers the best record of the torsion deformity to be a measure in degrees made by some instrument of the type described by Schulthess. The model shown in the illustration (Fig. 190) is one which has the advantage of being useful for a variety of purposes. Thus, it may be used to record the difference in the height of the shoulders or of the scapulæ; the inclination of the pelvis may be measured by the external method of Engelhard. Engelhard,* measures the inclination of the pelvis by ascertaining the deviation from the horizontal of a line joining the antero- and posterosuperior iliac spines. He found the average inclination of this line in a considerable number of observations to be 32°. Caliper arms are furnished with this instrument by which this inclination is very easily measured. The obliquity of the pelvis as shown by the difference in height of the two anterosuperior spines is also thus conveniently measured and we have in this a check in compensating for the shortness of a leg by means of a high shoe.

Diagnosis.—The distinctions between postural and structural scoliosis have been already discussed; there remains to be emphasized the necessity of ascribing to an admitted structural scoliosis its true nature from the viewpoint of etiology and pathology and, as well, to speak of those conditions in which lateral deviation of the spine is a symptom. The chief one of these is tuberculous disease of the spine. Whereas this condition is easily recognized once a pronounced knuckle, or gibbus, is present, it sometimes happens that quite marked lateral deviation is to be seen before the knuckle has formed; the writer has twice seen patients of adolescent age in whom a marked lateral deviation of the upper dorsal segment of the spine was observed as a symptom of early disease of the lower cervical segment of tuberculous character. Tuberculous disease of the spine has as its most marked characteristics, stiffness of the spine, referred pains in shoulders, chest, abdomen or thighs, according to the location of the lesion in the spine; alteration of the gait and the constitutional disturbances peculiar to tuberculosis. Torsion deformity of the chest and spine as above described is not to be found. The same thing is to be said of the other diseases of the spine which are frequently observed: For example, malignant tumors either primary or secondary, arthritis of the spine whether of the infectious or hypertrophic type.

Prognosis.—Just as in the case of postural curvature, it is often said of structural scoliosis that it will be outgrown without special treatment directed to it; erroneous in respect of the postural curve, there is even less likelihood of this in the case of the structural form, as might be imagined. On the contrary, while structural scoliosis frequently comes to a standstill independent of any treatment, there is in the writer's opinion, grave reason to doubt whether any unquestionably structural case is ever *completely* cured by any treatment whatsoever. As the chairman of the Scoliosis Commission of the American Orthopedic Association, which for three years devoted itself to a study of this question, and as one who has for years interested himself in it, it is somewhat disheartening to be compelled to make this pessimistic statement. This is especially true in view of the fact that Abbott stated in 1911–1912 that the cure of "fixed lateral curvature of the spine" was not difficult and presented in his two papers* numerous illustrations of cases which were of very severe types and cured in comparatively short periods of time, by his method. Although his method has been conscientiously followed by a number of experienced and competent men, they have not been able to report similar results; among orthopedic surgeons generally the method may be said to be discredited, at the present day.

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* Zeitschr. f. Orth. Chir., Bd. xxvii (1910).

Even though this must be said in the interest of reliability in statement, it is not to be assumed that there is nothing to be done for cases of structural scoliosis. On the contrary, not only can the majority of them be materially helped by treatment, if properly directed before the deformity has become fixed and before the patient has become a fully developed adult, and if undertaken in time and with thoroughness, but in the absence of such care it may be predicted as almost inevitable that further deterioration and increase of deformity will take place. That patients with considerable degrees of this deformity do not usually attain old age has already been adverted to and this alone should constitute a sufficient reason for energetic treatment at a time when definite accomplishment may be looked for even though we are compelled to acknowledge a real cure as unattainable at the present time. With the institution of proper measures of treatment, pursued with energy, skill and sufficiently long continued and in the presence of real coöperation on the part of the patient, we may hope for definite improvement in the appearance of the figure; if the deformity has not yet assumed a very high grade, especially with regard to torsion, we may expect definite reduction in the measures of deformity in patients who have not yet completed their body growth. When torsion deformity exceeds 10°, it can usually not be much diminished by treatment; this, at least, is the experience of the writer. The lateral deviation can nearly always be sensibly diminished. No decision concerning these accomplishments is of value, however, unless based upon careful measurements made after treatment has been abandoned for a considerable time. The decision should certainly not rest simply upon personal impression or upon photographs made by a method not standardized or simply posed for the occasion in a position which the patient cannot possibly maintain permanently. That we must be honest with ourselves stands to reason.

Prophylaxis.—Much has been writen on the relation of school and other occupations to the causation of scoliosis. It will be apparent that the maintenance of a vigorous musculature and a normal posture is of great importance in preventing the development of any postural fault of the trunk. If the development of structural deformity of the spine depends upon an added element concerned with the texture of the vertebræ and the ribs, whose precise nature is not understood, we shall have to acknowledge that these considerations will not avail us greatly in preventing structural scoliosis and we shall not be surprised to learn that it occurs very often in children whose surroundings both at home and at school are beyond criticism. That it occurs prevailingly in children of delicate muscular fiber, just before or during the early period of puberty is, however, quite certain; we should therefore be able to accomplish something in the way of prevention by a careful observation of children at this period, with a view to developing a vigorous musculature and a correct posture. What has been said concerning the hygiene of the child, in connection with postural scoliosis, is of added importance in respect of the structural form.

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* N. Y. Med. Journ. (Apr. 27, 1911 and June 24, 1912).

Treatment.—For many years the one remedy whose efficacy was recognized for the correction of postural faults of the spine was gymnastics. It would seem to have acquired its reputation by reason of the fact that it is of unquestioned value in purely postural scoliosis, that postural and structural scoliosis have for so long a time been erroneously grouped together and, finally, because even in frankly structural cases of not too severe degree it is possible by gymnastic methods alone to train the patient into a better posture and thereby to improve the appearance of the figure in a manner quite obvious. If, however, as seems absolutely necessary in the interest of real progress, we separate the postural from the structural cases and if we require as evidence of success in treatment a definite change for the better in measurements carefully made and compared without bias, we shall have to say that such evidence is not at hand and the burden of proof must rest upon him who says that by gymnastic methods alone the elements of bony deformity can be influenced to a degree capable of satisfactory demonstration. By no one has accuracy in the measurement of the elements of deformity and in the application of treatment by gymnastics and corrective movements been exceeded in comparison with Schulthess, of Zurich. Yet in the numerous publications which have come from him and his associates we shall seek in vain for convincing evidence of the kind which we require; nor is it to be found elsewhere. This is true even of the mildest cases *of unquestionably structural character.* We shall have to assume, therefore, that however valuable and indispensable as a part of the treatment of all scoliosis, gymnastics may not be relied upon as the means toward real correction in structural cases.

The effort has been made, for many years, to secure real improvement by methods akin to those which have been employed in the case of bony deformities of the extremities; by the use, therefore, of corrective apparatus, either in the form of braces which could be removed and applied at will, or since the time of L. A. Sayre, by the use of the plaster-of-paris jacket. It is indeed seductive to hold the condition analogous to club foot and to set up a theorem in accordance therewith; first correct the deformity by forceful means, if necessary, and to excess. Then by mechanical support hold this over-correction, the spine being permitted to bear weight, so that by the process of functional transformation spoken of as "Wolff's Law" the bones become actually changed in form. Finally, by means of gymnastic cultivation, bring the muscular strength up to the point where the newly acquired normal posture becomes permanent. This is a complete and entirely rational program; for success it depends, however, upon the possibility of solving the first term of the problem, namely, the correction or over-correction of the deformity in its elements. Wullstein* was able to produce unquestionable structural scoliosis in young dogs by holding them in scoliotic postures for long periods by means of a harness; should it not be possible to reverse this process in young individuals who have acquired structural scoliosis? He made the effort, using high degrees of force, and his example has been followed by many others with as many modifications of method. Up to the present time, evidence is lacking however, that it has been possible for anyone to produce complete correction of the elements of deformity in any but the mildest degrees of structural scoliosis; of enduring over-correction there has been no question at all. Abbott† believed that he had been able to over-correct; that he had succeeded in transferring the torsion hump from the one side to the other by using force in the flexed position of the spine. This has been shown to be an error of observation.‡ Owing to the elasticity of the ribs, it is easily possible in the flexed position of the trunk to twist them around the spine so that a marked bulge appears on the side of the concavity. It has not been possible to make the vertebral bodies twist around with the ribs, however, and the apparent over-correction cannot therefore be maintained. Unless the deformity has become greatly "fixed," as in the older patients, it is possible to diminish the lateral deviation and at least the appearance of torsion deformity by placing the patient in the horizontal position, either prone or supine, and still more by suspending the body from the head. The temptation is great to strive for more correction in these positions by using lateral force and holding what has been thus gained by the application of plaster jackets. Many methods have been used. There can be no question that the appearance in most of these jackets is that of great improvement. Unfortunately, we shall seek in vain for evidence that the torsion element has been considerably diminished by them, even after the jackets have been worn for long periods; and the torsion element is obviously the more important to correct, as it is the more difficult. Indeed it seems inevitable, when much force is used to correct the lateral bend, that the torsion deformity of the ribs will be made greater instead of less. This was shown by Schreger,§ in 1810, who showed that lateral pressure on the ribs of the convex side must result in increase of the backward bulge. Nevertheless this is a method of correction which we see recommended even at the present day.* The results of the various methods striving for the correction of the elements of the deformity by the use of force and retention by means of plaster jackets can therefore, in the view of the writer, not be looked upon as constituting great progress since the time when Sayre introduced the plaster jacket. In the course of a paper on this subject, written in 1912, the writer said: †

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* Zeitsch. f. Orth. Chir., Bd. x, p. 177 *et seq.*

† *Loc. cit.*

‡ Report of the Scoliosis Committee of the American Orthopedic Ass. Am. Journ. Orth. Surg., xiv (1916).

§ Quoted by Schulthess in Handb. d. Orth. Chir. Joachimsthal, Bd. i, Theil 2, p. 1039.

In striving for correction by forcible means with retention in jackets or apparatus we must observe correct mechanical principles.

1. The lateral bending of the spine has always been an alluring object for attack. Its effect on the distortion of the figure is so evident and it seems so easy to overcome by stretching in suspension and by lateral pressure or pull that in spite of repeated remonstrances we find methods recommended even at the present day with this objectionable factor of lateral pressure incorporated. It has constituted a most important advance to recognize in the torsion deformity of spine and chest the most important element of distortion as well as the most difficult one to dispose of. The diagonal diameter of the chest from dorsal rotation hump in back to pectoral rotation hump in front is lengthened; that diameter of the chest of the opposite sides is shortened (Fig. 186). Corrective pressure in the chest must be *entirely* and *exclusively* in the line of the lengthened diagonal in order to be corrective and not positively harmful. Purely or partly lateral pressure or pull, even though apparently corrective of lateral bend, must increase the torsion humps, lengthen the too long diameter and shorten the one which is already too short. The patient who has suffered this has been further damaged and not benefited.

2. Corrective force must be applied directly at the sites of maximum torsion deformity. Attempts to manage the dorsal or lumbar segment as a whole, result in apparent correction whose seat is really in the normal or comparatively normal parts of the spine. It will, therefore, be transitory since the essential elements of deformity are not markedly influenced.

Trial with the methods here advocated was abandoned for the time by reason of the remarkable results claimed for the Abbott method. Aside from the disappointing reports of the Committee on the Treatment of Structural Scoliosis which have been referred to, the efforts of the writer with the Abbott method were subjected to careful test by mensuration, according to the method above described and the views expressed in his former paper fully corroborated.‡ In accordance with what has been said, the writer's efforts at the correction of structural scoliosis by means of force consist at

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* Rich, Journ. A. M. A. (December, 1911); Lovett, "Lateral curvature of the spine" (1916), p. 162; Whitman, "Orthopedic Surgery" (1919), p. 209.

† Freiberg, Am. Journ. Orth. Surg. (1912), pp. 9–10.

‡ Am. J. Orth. Surg. (1913), p. 29 *et seq.*

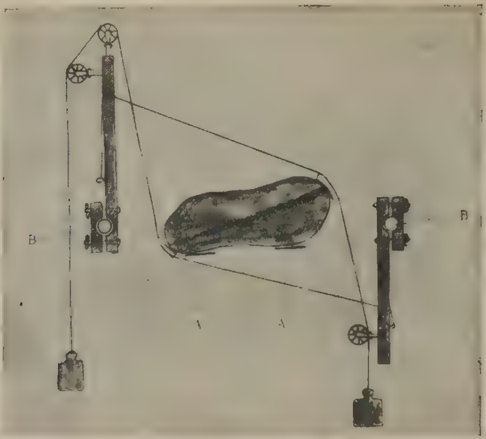

Fig. 198.—Cross-section showing the method of applying purely diagonal pressure to the scoliotic chest, with the patient horizontal. At *b*, *b*, the gas-pipe frame is seen in cross-section; *a*, *a*, represent the webbing straps upon which the patient is supported.

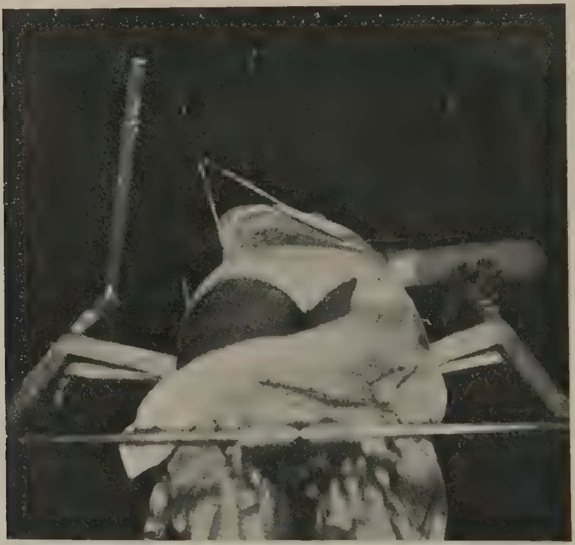

Fig. 199.—The jacket has been completed. The right dorsal rotation hump has been overcorrected as shown by the backward bulge of the left chest. There was torsion deformity of the right side amounting to 13°.

the present time in the application of jackets either in the horizontal position or in the vertical with traction on the head; force is applied, however, not at all with a view to overcoming the lateral bend but entirely in the long diagonal of the chest. The figures show the manner in which the torsion element is attacked (Figs. 198, 199). Although the primary results seem encouraging, much more requires to be done before the true and therefore enduring effects of this method may be evaluated. To be truly corrective, the jackets must be made quite uncomfortable to the patients and it seems clear that they must be worn for long periods. Lasting correction of the elements of bony deformity will not be attained with much less than a year of treatment by corrective plaster jackets; it is likely that this will most often not be long enough. It will be apparent that during this long time, as the result of the immobility of the trunk and pressure upon the muscle masses necessarily attendant, a marked degree of muscle atrophy will take place. It may, therefore, be stated in definite terms, that whatever method has been used for correction by means of jackets or corrective appliances, more harm than good will have been accomplished by them unless a vigorous and long continued course of gymnastic cultivation of the muscles be pursued, together with the use of removable retentive apparatus. It were better, in fact that no attempt at this kind of corrective treatment be made, unless the opportunity for effective after-treatment by gymnastics be at hand. The correction will, otherwise, not only be lost but there is good reason to believe that by reason of the loss of muscular control the deformity will finally be greater than if nothing had been done. Efficient gymnastic treatment is therefore to be regarded as a *sine qua non;* if either forcible correction by means of jackets or gymnastics is to be employed to the exclusion of the other, the jacket treatment had better be omitted.

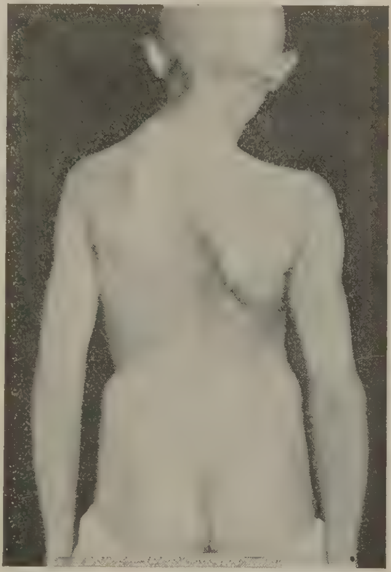
Fig. 200.—Cervical dorsal structural scoliosis to the left from infantile paralysis in a boy of 12.

Teschner* has described a system of exercise treatment for scoliosis which may be made to give excellent results, if carefully carried out and within the limits set by the nature of the deformity. Space forbids description in detail of this treatment; its purpose is to develop the strength and muscular control of the whole body. and not simply of the trunk. By means of "heavy work," he aims to bring the trunk muscles to the greatest degree of development consistent with safety. None of the exercises is offered as having especial corrective effect upon the deformity. This is not true of a number of other systems, but there is no tangible evidence at the present time that any exercise treatment, alone, has the slightest lasting corrective influence upon structural scoliosis. The place of gymnastics in the treatment of structural scoliosis is, therefore, as means of restoring and improving the muscular development after corrective efforts have been made by the application of forcible jackets or apparatus or for the purpose of building up the patient's muscular control when these have not been used; not in the latter case with the hope of thereby diminishing the degree of deformity, however, but with the expectation of holding further deterioration in check and of helping the patient to a better state of general vigor. When exercises are to be used with either of these objects in view, the same methods may be followed as have been recommended for the treatment of postural scoliosis.

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* Annals of Surgery (August, 1895).

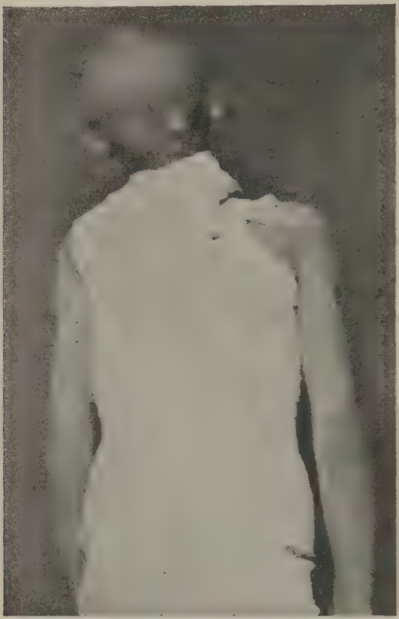

FIG. 201.—Posterior. The same patient after the application of a corrective plaster jacket with neck support to control the cervical segment.

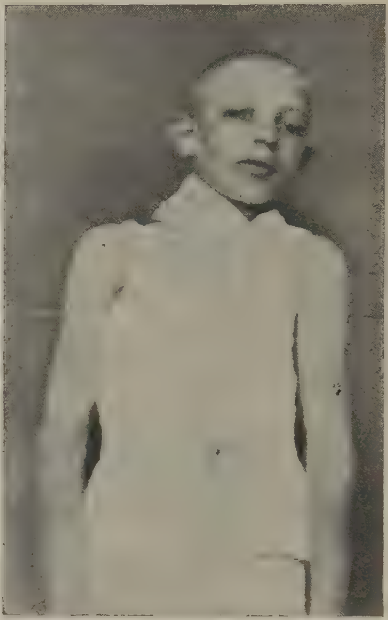

FIG. 202.—Anterior. The same patient after the application of a corrective plaster jacket with neck support to control the cervical segment.

The failure of widely discussed methods of forcible correction to fulfill the hopes which were entertained for them and the unsatisfactory prognosis which must still be given to the patient with structural scoliosis, have served to bring about efforts to ameliorate the condition by operative methods. On the part of Sauerbruch and others the attempt has been made to influence the spine by means of operations on the thoracic cage, in the hope that, inasmuch as scoliosis often follows empyema and operations done upon the ribs for it, a reversal of such effect might be thus secured. The principle was really originally invoked by Hoke.* It cannot be said to have had any beneficial effect, either in his hands or those of others. McKenzie, Forbes, Hibbs, Whitman, Kleinberg and others† have sought to supplement the correction of the deformity when it has been accomplished so far as possible by postural and mechanical means, through operative procedures. These operations have had as their purpose the stiffening of the segment of the spine involved in the scoliotic curve in a manner similar to that employed in spinal tuberculosis. The operations which have been done have consisted of modifications of the fusion procedure of Hibbs, on the one hand, or the grafting of bone into the involved area, as originated by Albee. A combination of both methods has also been used. The reports of these operations are too recent to permit of final judgment on the part of their own protagonists. It seems possible to say this much, at the present time, however:

1. The essential problem of scoliosis, which is the veritable and approximately thorough correction of the vertebral deformity, has been brought no nearer solution.
2. The most which may be expected of any operative procedure which has thus far been reported, is that it may make permanent such partial correction as has been produced antecedently, by means of permanently stiffening the spine.
3. All of the operations thus far proposed, and looking to this end, are severe involving no little shock and suffering and prolonged after-care.
4. Even if the results which have been exhibited are to be regarded as permanent, they seem to the writer to be an inadequate reward for what the patient is required to endure.
5. It has still to be shown that a considerable portion of the spine may be permanently immobilized in this manner, without detriment to the future health of the patient. One might bear in mind, in this connection, the frequency with which patients whose spines were stiffened by ankylosing arthritis, have succumbed to pulmonary tuberculosis.
6. A very conservative attitude toward the operative methods in the treatment of structural scoliosis, seems justified by these considerations.

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* Am. Journ. Orth. Surg., 1903, p. 168.

† Forbes, Am. Journ. Orth. Surg., 1920, p. 357.

CHAPTER CXV

THE SHOULDER JOINT

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SUBACROMIAL BURSITIS

Synonyms.—Subdeltoid bursitis; peri-arthritis-humero-scapularis.

Although not common in childhood, this condition often simulates one or the other of the numerous pathological conditions within or about the shoulder joint so that for the sake of differential diagnosis, if not for other reasons, a discussion of the clinical features of subdeltoid bursitis, within the scope of this work, does not seem to be out of place.

The bursa known as subdeltoid bursa extends from the upper third of the deltoid muscle upward under the lower surface of the acromion process separating the greater tuberosity of the humerus from the underlying muscle, and the head of the humerus from both the acromion process and the coraco-acromial ligament. When this bursa is filled with effusions they point toward the anterior edge of the deltoid muscle and are then most easily reached at this point.

The function of the subdeltoid bursa is that of a mechanical gliding apparatus established between the humeral head and the overlying structures. This gliding mechanism is indispensable to the abduction and rotation movement of the shoulder. In abduction the bursa dives under the acromion and again becomes more prominent when the arm is adducted. Corresponding changes in the configuration of this bursal sac occur in rotatory movements of the arm.

The bursa is subject to inflammatory changes of various types. Acute inflammation of the sac has been observed with hemorrhagic, serous or purulent contents. But of especial interest are the chronic inflammatory conditions about the bursa. They cause typical chronic disabilities of the shoulder joint, and the study of this syndrome is of special interest from the viewpoint of differentiation between other disabilities occurring in and about the shoulder joint.

In the first place it must be considered that traumatism of the shoulder joint or of the surrounding muscles and tendons often involves the subdeltoid bursa at the same time, causing a complexity of symptoms into which only careful analysis can bring a clear conception of the underlying pathological condition.

We know since the observations of Codman that the tissues most often involved in subdeltoid bursitis are the floor of the bursa as well as the adjacent tendons. Of the latter structures it is the tendon of the supraspinatus muscle which shows most frequent changes. Tears in this muscle occur and are followed by hemorrhage, and result in formation of scar tissue, infiltrating the tendon. Under certain conditions, lime salts are accumulated in this scar tissue forming extra-bursal deposits in the tendon.

In cases in which there is no lime salt incrustation of the tendon—and these cases form a large majority—there is no positive *x*-ray evidence. In those, however, in which a deposit of lime salts exists, the *x*-ray shows a quite distinct shadow which appears laterally to the greater tuberosity of the humerus and is distinctly separated from the bone. The *x*-ray findings or their absence, however, seem to bear little relation to the presence or degree of clinical symptoms.

Inasmuch as the subjective symptoms are not by any means bound to the deposit of lime salts but are rather due to inflammatory changes around the sac and to the involvement of the tendons, it is the clinical examination and especially the functional test of the humerus which forms the basis of the diagnosis.

The patient complains of persistent pain located over the deltoid muscle. Pressure over this muscle below the tip of the acromion usually elicits distinct pain. Upon certain motions this pain over the shoulder joint becomes distinctly aggravated. These motions are abduction and outward rotation. In the position of abduction the tenderness on pressure over the deltoid muscles occasionally disappears, which is explained by the disappearance of the inflamed bursal sac underlying the overhanging acromion which takes place in this position.

Radiation of pain is very frequently observed. It usually follows the distribution of the internal cutaneous nerve occasionally involving the elbow joint.

Due to the tenderness of the deltoid region and the exquisite painfulness of certain motions, one will find the joint usually in a state of contracture. Active abduction is usually totally lost. Passive abduction is possible only to a limited extent. Outward rotation is also very greatly limited if not entirely abolished while inward rotation is comparatively free.

Otherwise the configuration of the shoulder is little changed except in those cases in which there is effusion in the bursal sac. Deltoid atrophy is usually insignificant.

Differential Diagnosis.—The most important point in examining the motion of the shoulder joint is that there is not complete arrest of motion but there is always some motion even if limited, in the direction of forward flexion, adduction, and inward rotation. This is the most important point in regard to differential diagnosis because it proves the lesion to be extra- and not intra-articular. This distinction between extra- and intra-articular lesion of the shoulder joint from clinical observation is the first point which should be established.

If an effusion exists in the shoulder, the relation of the shoulder blade to the humerus is, as has been shown by Lange, quite typical and characteristic. In true effusion of the joint, the latter maintains a pathognomonic position of abduction between 25° and 30°, and slight forward flexion. This position is being maintained throughout all motions carried out by the humerus, or, in other words, the joint in intra-articular lesion appears consistently fixed in this position and all movements of the humerus are therefore accompanied by such rotatory or translatory motions of the scapula as are necessary to maintain the pathognomonic position.

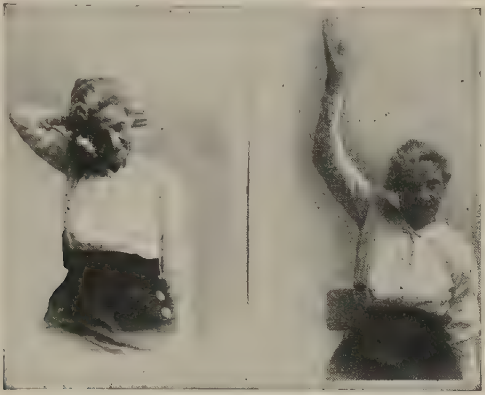
So, when the arm is let down to the side of the body, the shoulder blade masks it position of 25° abduction and slight forward flexion by a rotation about its sagittal axis, which can be noted by the fact that the vertebral border of the scapula is no longer parallel to the line of the spinous processes, but its lower angle is approached to the line while the upper is farther removed from it, giving the inner border a slightly downward and inward slant.

If, on the other hand, no effusion or inflammatory condition exists in the shoulder joint, partial motion at least in a certain direction is possible so that the scapula maintains its normal position throughout a certain range of motion without changing its relation to the spine. In children there occur various sprains and tears in the neighborhood of the humeral head which may resemble clinically lesions of the bursa. Of the tendons most commonly involved there are to be mentioned the latissimus dorsi and pectoralis major. Both conditions cause adduction contracture but the seat of the injury can readily be located by the distinct tenderness which exists at the point of insertion of the tendon into the humeral shaft.

Sometimes diagnostic difficulties arise from nerve symptoms of the brachial plexus which follow distensions or tears of the shoulder joint capsule. These tears are regularly accompanied by extravasation of blood and serum and are followed by formation of scar tissue which surrounds the neighboring nerve trunks. The result of these scar adhesions are neuritic symptoms of unusually wide radiation into arm, forearm, and sometimes into the finger. The portion of the capsule most frequently involved in tears or strains is the lower and inferior or axillary part. After reduction of dislocations of the subacromial or subglenoid type, these parts of the capsule often remain tender, and for this reason abduction of the arm is instinctively resisted by contracture of the adductors. The local examinations of the joint capsules in the armpit together with the history will facilitate the diagnosis. The question of adduction contracture in birth palsy will be the subject of discussion in a later chapter and need not be touched upon on this occasion beyond emphasizing the fact that the history and the evidence of paralysis will weigh considerable in making the diagnosis.

The condition of the deltoid muscle itself is of differential value inasmuch as it shows little atrophy in subdeltoid bursitis, whereas in traumatic conditions of the shoulder joint or in inflammations the atrophy of this muscle is usually very pronounced.

Treatment.—Acute cases yield almost regularly to fixation in splint or plaster-of-paris cast. As time goes on, however, the prognosis of subdeltoid bursitis becomes more uncertain. Most of the chronic cases respond to manipulations carried out under anesthesia with the arm placed in abduction and outward rotation. Forcible breaking up of adhesions is not to be recommended and it is preferable to stretch the muscles gradually and to obtain the desired position of abduction and outward rotation by steps rather than immediately, wherever any obstacle should present itself to the complete correction under the anesthesia when the muscles are relaxed.



Figs. 203, 204.—Subdeltoid bursitis. Result of conservative treatment.

Fig. 205.—Subdeltoid bursitis. *X*-ray showing calcareous deposit.

According to Brickner's advice, the position of abduction and outward rotation may be obtained also when the patient is in recumbent position by having the afflicted arm fastened to the pillow, in which case the body-weight acts as extension and gradually forces the arm into the desired position.

The after-treatment is to begin a few days after manipulation and consists in massage and application of heat. The splint which had been applied at operation is then gradually lowered until in the course of several weeks or even months it may be entirely dispensed with. A number of chronic cases resist manipulative procedures and do not yield to conservative measures. These cases, according to some observers, as well as the type of cases showing definite deposits of lime salts in the supraspinatus tendon in the x-ray picture, require open operation.

The incision is best made over the lateral aspect of the deltoid muscle from the acromion downward proceeding bluntly through the muscle fibers. Once the under side of the muscle is reached, the roof of the bursa is incised and the edge is secured by fine forceps. In similar manner one proceeds through the floor of the bursa under which the lime salts deposit is found imbedded into the tendon of the supraspinatus muscles. This deposit is then carefully removed and the floor as well as the roof of the bursa is sutured and the wound is closed.

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HABITUAL DISLOCATION OF THE SHOULDER

Habitual dislocation of the shoulder is occasionally seen in children or younger individuals. It follows injuries which produce relaxation of the capsule of the shoulder joint. Sometimes it is a tear in the rotatory muscles of the arm which is responsible for the condition. Sometimes muscular atrophy following paralysis of the deltoid may be the cause of habitual dislocation. But in the majority of cases it is injury to the capsule itself, especially in its inferior portion, which produces the pathological changes that underly the disability. These changes consist essentially in a more or less marked degree of relaxation of the capsule as the result of repeated stresses and strains, or, more often, in incomplete tears and rents in the capsule. Occasionally, recurrent dislocation is found as sequela to traumatic dislocation.

The mechanics of this condition have been studied extensively by T. T. Turner. He believes that extreme motions of the shoulder tend to force the humeral head out of the socket but are ordinarily checked by the tightening of the capsule and its reinforcing ligamentous and tendinous structures. These extreme motions are particularly those of abduction and outward rotation in which the axillary portion of the capsule is under stress and may give way by a rent in its structure. Occasionally this rent is big enough to allow the head to pass, and complete dislocation of the humerus is then established. But more often these rents are incomplete and cicatrize finally together leaving an inelastic scar which prevents the capsule from tightening up and makes the subsequent escape of the head from the socket so much easier.

The ligamentous capsule of the shoulder joint is a rather feeble structure. The shoulder owes its stability mainly to the muscular apparatus surrounding the capsule and especially to the subscapularis, supraspinatus, infraspinatus, and teres minor muscles. The only portion of the joint capsule which is not guarded by muscular insertions is the space between the origin of the long head of the triceps at the lower edge of the glenoid fossa below, and the insertion of the subscapularis tendon above. It is in this portion where the capsule is most frequently torn and where as the result of primary tears a relaxation of this portion of the capsule occurs with subsequent recurrent dislocations.

Treatment.—Patients seen immediately after displacement may be treated successfully by fixation followed by massage and exercise of the atrophic muscles. Cases in which the dislocation is of more or less recent date and in which it has recurred only a few times and at long intervals, may yield to this treatment. Fixation should be carried out in adduction, and the shoulder should be supported by a shoulder-cap of canvas or leather fitting closely to shoulder and upper arm.

Most cases of long standing, however, and those with frequent recurrences of the dislocation call for operative methods. The following methods have been advised:

1. Capsulorrhaphy.—In abduction of the arm an incision is made from the coracoid process downward. The deltoid and pectoralis muscles are separated, the humerus is rotated outward bringing into view the insertion of the subscapularis tendon which is now divided. The capsule being thus laid bare is incised longitudinally and then tightened by overlapping the ends of the incision and suturing them tightly with No. 3 chromicised catgut.

2. Burrell and Lovett use an incision from the coracoid process downward following the line of the cephalic vein. One proceeds bluntly between the deltoid and pectoralis muscles until the coracobrachialis and the short head of the biceps come into view in the upper end of the wound and the insertion of the pectoralis major at the lower angle. The head and neck of the humerus are then exposed by a blunt dissection. The antero-internal aspect of the capsule is then exposed, the subscapularis tendon is divided, and the muscle retracted. The arm being in abduction of 25°, the relaxed part of the anterior and inferior portions of the capsule is now grasped in a fold with hemostates. Catgut sutures are now inserted through the fold and then an elliptical piece 1 in. long and ½ in. wide is dissected between the sutures already placed.

According to T. T. Thomas, the capsule may be exposed by three routes; a delto-pectoral or external route, an antero-axillary, between coracobrachialis and the axillary vessels, and, finally, a postero-axillary route posterior to the axillary vessels. He gives the preference to the postero-axillary incision which gives exposure just where the changes in the joint lesion are best detected and where no muscles have to be divided.

3. Clairmont-Ehrlich advocated an operative procedure by which a small flap is formed from the posterior portion of the deltoid and is carried around the neck of the femur forward. It is then fastened to the edge of the anterior portion of the deltoid muscle from a second incision made over the anterior aspect of the shoulder joint. Such plastic operations are considered by some (Sever) as unnecessary in view of the fact that the trouble is due to relaxation of the capsule and that capsule pleating operations are giving uniformly good results. Some English observers, however (Ollerenshaw), prefer the Clairmont-Ehrlich method of muscle plasty.

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BIRTH PALSY

Synonym.—Obstetrical paralysis.

The fact that paralysis of the upper extremity at times follows upon the delivery of a child in the presence of certain obstetrical difficulties necessitating forcible manipulations, has been known for over 100 years. As early as 1872 it was recognized by Duchenne as a laceration process of the shoulder following forcible delivery. Erb furnished a classical description of the condition, which he recognized as a lesion of the brachial plexus. It was first explained by the older observers as a paralysis due to direct pressure by the forceps upon the plexus cords.

The investigations of the last decade, however, have brought forth a great deal of information bearing upon the pathogenesis of this deformity, and it is now generally assumed that the seat of the nerve injury is in the spinal roots of the brachial plexus involving particularly the fifth and sixth cervical roots. This latter distribution is by far the most common and represents the Duchenne-Erb or upper arm type of birth palsy.

Numerous experimental facts have been reported concerning the influence of traction or pressure upon the roots of the plexus. It seems that there is little danger to the plexus from the pressure of the clavicle itself, but if the clavicle is broken the upper plexus roots break much easier than they do when the clavicle is intact. The experimenters agree regarding the fact that the burden of the strain put upon the brachial plexus by pulling the arm downward falls upon the fifth and sixth cervical roots while the tension upon the lower roots of the plexus is not nearly as great. This is in substance the basis of the neurogenetic theory of birth palsy. But this theory, on the other hand, has found many ardent opponents who maintain that the seat of the injury is in the shoulder joint itself. Lange considers most cases of birth palsy as due to laceration of the shoulder joint and Vulpius attributes the condition to a separation of the humeral epiphysis. In this country, T. T. Thomas is foremost among those who consider injuries of the shoulder joint as the primary and most essential cause of the trouble. He believes that when this factor is removed one need not be concerned about the paralysis and that the joint injury is primary and the paralysis secondary, the latter being caused merely by the inclusion of the branches of the brachial plexus in the axillary exudate consequent upon joint injury. The disappearance of the paralysis which is almost regular in the Duchenne-Erb or so-called upper arm type is advanced by Turner as positive proof of the absence of any rupture of the nerve roots, and it is also pointed out by him that in obstetrical paralysis even of the upper arm type there is a severe if not complete paralysis of the whole limb and the damage is not merely confined to deltoid and supraspinatus muscles. Thomas further believes that the traction of the forceps cannot be responsible for the injury inasmuch as there is a number of cases of normal delivery appearing in the series of birth palsies; (in the series reported by J. J. Thomas and Sever, 32 of 471 cases had normal birth). Finally, the experiments of T. T. Thomas show that even extreme traction of the head by the forceps is not capable of producing any visible rupture at any point of the plexus. Equal discrepancies are being found in the operative reports. A. S. Taylor states that he finds operatively ruptures of the fifth and sixth cervical roots while others have failed to note nerve lesion at operation. But even the positive operative findings do not entirely silence the arguments of the opponents of the neurogenetic theory, namely, that the shoulder contractures which follow birth palsy are proof of an existing articular or periarticular lesion. If the condition is entirely a neurogenetic one, why is it that a flail joint does not develop such as we see developed following deltoid paralysis in anterior poliomyelitis? Furthermore, it is hard to conceive that the posterior dislocation as seen in birth palsy is entirely a paralytic deformity inasmuch as we do not see the like in true paralytic conditions.

In view of these discrepancies, in clinical and operative observations, one is compelled to assume that neither of the two theories can be accepted to the entire exclusion of the other. Indeed, there seems to be no good reason why a gross lesion of the shoulder joint might not in a number of cases be combined with injuries of the nerve roots. The weight of common opinion at the present time undoubtedly points to nerve injuries as a cause of birth palsy, but joint injuries should at least in a certain class of cases be considered as concomitant complications.

Clinical Pathology.—1. Upper Arm Type.—The deformity is noticed very shortly after birth, usually within the very first days of life. The affected arm hangs limply at the side of the body. The elbow is usually held in slight flexion with the forearm pronated. During the first week there may be some swelling and tenderness in the region of the deltoid muscle and of the shoulder in general. This, however, soon subsides, and there remains only the paralysis of the deltoid and supraspinatus muscles to which is added occasionally paralysis of the biceps, coracobrachialis, infraspinatus, and supinator longus muscles. Together with the inability of abducting the arm which is usually noted within the first month, a contracture develops on the basis of the unopposed action of the adductor and inward rotator muscles of the shoulder. The inward rotation of the shoulder is produced mainly by the subscapularis, while in the adduction contracture, the pectoralis major and the latissimus dorsi muscles are most prominently engaged.

There is no doubt that some cases go on to complete recovery without the establishment of a residual contracture, but in most cases the contracture in adduction and inward rotation remains as a residual deformity after the paralysis has disappeared. In the upper arm type of birth palsy there is only a small minority of cases in which no nerve repair takes place and the paralysis remains permanent.

In a small number of cases the contracture of the shoulder is complicated by subluxation or dislocation of the humerus. Cases of this kind have been advanced as arguments for those who consider the joint injuries as primary factors in birth palsy. If, on the other hand, this condition is to be explained as a result of muscular contraction following nerve lesion, it must be assumed that the contracted subscapularis muscle forces the head backward and that the relaxed posterior capsule of the joint as well as the relaxed tendon of the paralyzed supraspinatus muscle permits the head to leave the glenoid cavity in a posterior direction. The head then appears posteriorly and slightly below the acromion process. The latter is often hooked downward over the glenoid fossa forming a considerable obstacle to the replacement of the head.

2. The Whole Arm Type.—In a minority of cases of obstetrical paralysis, the lesion involves all the cords of the brachial plexus so that the paralysis appears distributed over the entire extremity. In these cases there exists also a paralysis of the flexors and extensors of the fingers and a decided atrophy of the intrinsic muscles of the hand. Here, the lesion involves not only the fifth and sixth cervical roots but also the lower cords of the plexus, especially those forming the ulnar and median nerves.

Regarding the contractures developing in this type of birth palsy, the adduction and inward rotation position is the same as in the upper arm type, but in addition to this there is also a distinct pronation contracture of the forearm and often a paralytic drop-wrist.

3. The Forearm or Klumpke's Type.—Mme. A. Klumpke described in 1885 a group of cases of birth palsy in which the involvement of the entire upper extremity was followed by the rather quick recovery of the muscles of the upper arm group while the lower segments of the plexus remained more or less paralyzed. This type involving the inferior roots of the plexus only has since been known as the Forearm or Klumpke's Type of Birth Palsy. Both in the whole arm type and in the lower arm type, sensory disturbances are found These are distributed mostly along the outer side of arm and forearm, the inner side being less involved owing to the distribution of the intercostohumeral nerves and the internal cutaneous nerves of the arm. In the inferior radicular type, one also finds regularly the oculopupillary symptoms of myosis and of retraction of the eye-ball. This latter type is the least frequent of the three groups and the prognosis in these cases is even less favorable than it is in the whole arm type.

Complications.—One of the frequent complications is fracture of the clavicle. The experiments show that this complication greatly facilitates rupture or tear of the plexus, especially of the lower cords.

Displacement of the humerus occurs in some cases and is usually posterior, the head of the humerus being felt below the acromion and upon the spine of the scapula. Many of the cases reported as congenital dislocation of the shoulder are really cases of birth palsy with secondary dislocation of the humerus. Cases of true congenital dislocation of the humerus are extremely rare. The only positive evidence of a true congenital dislocation of the shoulder is the finding of a missing or rudimentary glenoid cavity or a distinct malformation of the humeral head. Cases of this kind have been reported by Hoffa and Smith who found that a new articular facet had formed near the original glenoid fossa.

Prognosis.—It has already been mentioned that a small minority of cases recover almost completely within a period of three months. The majority of cases of the upper arm type recovers as far as the paralysis is concerned but retains a residual position of contracture in adduction and inward rotation. For the whole arm type, the prognosis is much less favorable, and for the lower arm type it is distinctly bad as regards restoration of function.

Treatment.—The treatment should begin at the earliest possible period and should consist in immobilization of the arm in abduction and outward rotation by means of a plaster cast or a removable splint. A plaster cast may be applied for a month or two and then be superseded by the splint in order to carry out massage and passive motion.

Cases with severe contractures may require an anesthetic for the correction of position. In small children such procedures are usually sufficient. Older children, however, often require operative interference for the relief of the contracture. The best method advised for this purpose is that of Sever, the technique being as follows:

The incision is made from the acromion downward between deltoid and pectoralis major muscles. These are bluntly dissected and retracted outward and inward respectively. In order to gain access to the humeral head, the upper edge of the pectoralis insertion is incised. Then by outward rotation of the arm, the subscapularis tendon is located as it swings around the head of the humerus. A blunt director is then passed beneath it and the tendon is severed. This allows of complete outward rotation of the arm. The retracted muscles are then allowed to fall together and the wound is closed, the arm being put up in abduction and outward rotation. It is not necessary to open the capsule. In some cases the coracobrachialis muscle as well as the short head of the biceps are contracted and then their tendons must be severed also.

Other authors recommend the osteotomy of the humerus with subsequent outward rotation.

Treatment of the Dislocation.—In a number of cases the dislocated humerus may be reduced by bloodless manipulation. In the majority of cases, however, bony obstacles such as the hooking of the acromion prevent reduction and open operation is necessary. In order to hold the humeral head in place, Phelps advocated the arthrodesis of the shoulder joint.

Operation on the Plexus.—The indications for operative repair of the nerve roots are still somewhat indefinite because of the uncertainty of spontaneous nerve repair and because of the difficulty of deciding how long one should wait. Most observers agree that in severe cases one should not wait more than a few weeks. The operation as described by Taylor entails considerable technical difficulties.

The incision starts at the posterior border of the sternocleidomastoid muscle running obliquely downward to above the clavicle and from here outward. The subscapular vessels must be tied as well as the arteria transversalis colli. After the thickened and adherent deep fascia is divided, the nerve plexus is exposed. The injury involves usually the fifth and sixth cervical roots which are often imbedded in a mass of scar tissue and are extremely difficult to isolate. A number of operators have succeeded in bringing the nerve ends together by lateral suture and finally uniting the nerves end to end and sheathing the place of suture with Cargyle membrane. The consensus of opinion today points to the fact that ensheathing of nerve with any material save fat or muscle tissue is likely to produce adhesions and to interfere with the result. After operation, a plaster cast is applied which keeps the head and shoulder approximated to avoid tension upon the nerves. Sometimes it is necessary to divide the clavicle and the subclavian muscle in order to gain proper access to the plexus.

The reports on plexus operations are still uncertain. Thomas reports nine cases, three of which improved; among surviving seven, two died soon after operation. He performs his operation at ages from 6 to 12 months. Sharpe reported a series of 56 operations and failed to find a nerve lesion only in one case. He is an ardent advocate of early plexus operation, operating at the age of one month.

In the writer's series of 17 cases of birth palsy, 13 belonged to the upper arm type and four to the whole arm type. No case of lower arm type was observed.

Of the upper arm type cases difficult delivery or birth trauma is recorded in seven. Birth trauma was also recorded in all of the whole arm type cases. In two cases Sever's operation was carried out with good results. Two cases had dislocation of the humerus, one treated by open operation and the other by manipulative reduction. Of the upper arm type cases treated, all gave good results either by plaster fixation with or without manipulation or by Sever's operation. Of the four cases of whole arm type three gave fair results and one a poor result.

The case of posterior dislocation of the humerus treated by operation was a boy nine years old. The paralysis was noted within four days after birth and still involved the entire extremity at the age of 1½ years. He could flex the fingers and the wrist but could not extend the wrist. Neither could he abduct the arm in the shoulder. On admission, the patient had an adduction and inward rotation contracture of the shoulder, a posterior dislocation of the head of the humerus with hooking of the acromion, and a residual paralysis of the musculospiral nerve. There was no deltoid paralysis. The forearm was held in pronation contracture. In this case open reduction of the dislocated head was performed involving osteotomy of the acromion, and this

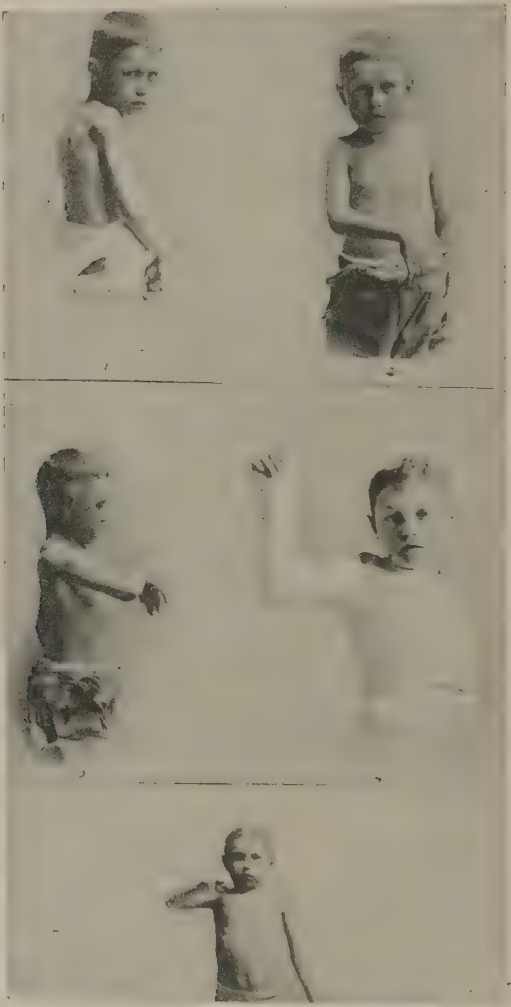

FIG. 206.—Birth palsy with posterior dislocation of shoulder. Operative correction. Whole arm type.

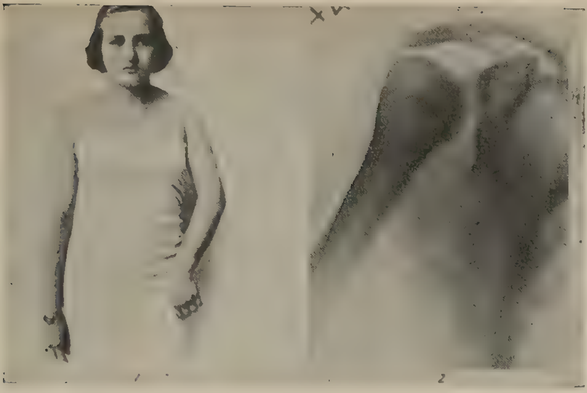

Fig. 207.—Birth palsy with posterior dislocation; whole arm type.

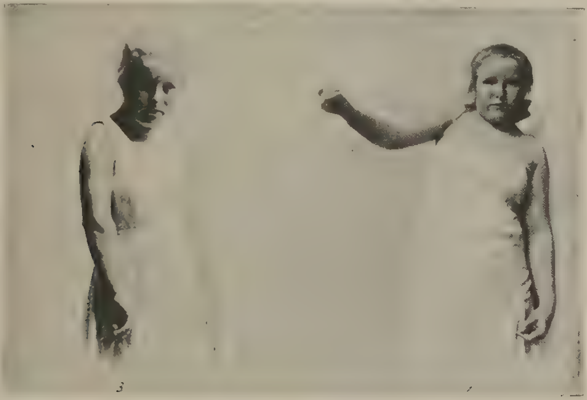

Fig. 208.—Birth palsy, whole arm type; before and after Sever's operation and pronator resection.

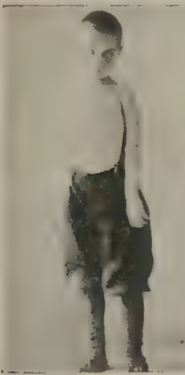

Fig. 209.—Birth palsy; whole arm type.

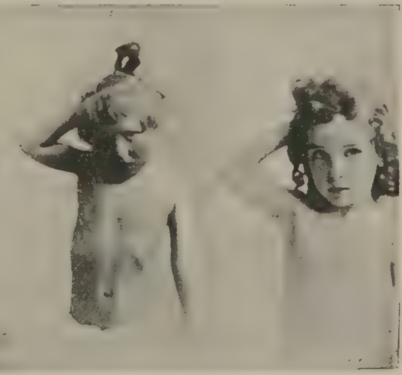

Fig. 210.—Birth palsies; upper arm type.

was followed by the stretching of the contracted subscapularis tendon (Fig. 206).

Another case of the whole arm type was a boy 4½ years old who had been seen off and on since he was eight weeks of age. At that time he had complete brachial palsy of the entire right upper extremity, and no motion in either shoulder, elbow or fingers. Six months later, his motion had gradually improved, flexion and extension appearing in hand and fingers, and some power of abduction in the shoulder. Treatment was consented to when the boy was 4½ years old. At this type the residual deformities had been well established. The flexion of the fingers was good, the extension rather poor, and the forearm was held in pronation contracture. Biceps and triceps muscles were active. Abduction of the arm was possible to the horizontal but was prevented beyond this point by contracture of the pectoralis and subscapularis muscles. There was also a dislocation forward of the head of the radius. The operative treatment consisted in Sever's operation for the relief of the contracture of the shoulder, in arthrodesis of the wrist for the relief of the drop-wrist, and, lastly, in the operative reduction of the dislocated head of the radius. Both of these cases are illustrative of the degree to which an initial paralysis is clearing up until it is narrowed down to a rather limited residual amount.

A girl 14 years old presented herself for treatment of deformity and disability of the right arm. In this case extensive paralysis of the right arm was noticed on the third day after birth. When seen the entire extremity was found shortened and there was extreme inward rotation in the shoulder together with adduction contracture, but there was distinct action of the deltoid muscle. The forearm was contracted in extreme pronation so that the palm was turned forward being rotated almost in a full circle. The operative treatment in this case consisted first in the relief of the extreme pronation contracture by open resection of the pronator radii teres and of the pronator quadratus. The adduction contracture of the shoulder was relieved by Sever's operation and the extension contracture of the elbow was finally relieved by a muscle plasty upon the triceps muscle. In the course of time this patient gained a very useful arm after she had been submitted to a rather extensive after-treatment consisting in massage, manipulation, and muscle drill.

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CONGENITAL ELEVATION OF THE SCAPULA

Synonym.—Sprengel's Deformity.

The deformity was described by Sprengel in 1891, who reported three cases ranging from one to seven years of age. Although early descriptions of this deformity are at hand, Sprengel's reports contain the first definite and accurate description of this condition.

In order to understand the pathology of the congenitally high or elevated scapula, the normal topographical relations between scapula and vertebral column must be considered. The upper border of the scapula reaches up to the second rib while the lower angle is situated at the level of the seventh and eighth rib. The root of the spine of the scapula at its vertebral border is situated at the level of the fourth rib and opposite the third thoracic spine. Normally, the scapula has a triangular shape with a long vertical and a short horizontal diameter.

The scapula in an embryo, 11 mm. in length, lies opposite the lower four cervical and the first and second dorsal vertebræ. In the course of development of the embryo, the scapula migrates downward. In a full-term human fetus the scapula still maintains a high position. In fact, the whole upper limb is in its original anlage, a cervical appendage retaining this position in fetal life. It is only with the beginning of extra-uterine life that mechanical influences come into play which change the position of the upper extremity. In congenital elevation of the scapula it is therefore to be assumed that the normal descensus of the scapula has been interrupted, it having failed to complete its downward move in the course of the embryonal development.

The scapula itself is changed in form having a long horizontal and a short vertical diameter, a change which denotes a reversal to lower types. Sometimes the upper outer angle of the bone is drawn out into a hook. A large percentage of cases also show fibrous or cartilaginous attachments of the scapula to the vertebral column. Sometimes there exist bony articulations between the scapula and the spine. These articulations run from the upper inner angle of the scapula or from its median border to one of the transverse processes of the cervical spine. The nature of these articulations is not clear. Some seem to think that they are an outgrowth of the spinous process while others believe that they are formed both by the spine and the scapula. Others again see in it an abnormal development of the suprascapular epiphysis such as exists as a narrow bridge of bone and is known as suprascapular bone in some of the lower vertebrates. There are other authors who see in these articulations merely degenerated muscle bands with centers of ossification appearing in them.

A second feature of this deformity are defects and anomalies in ribs and vertebræ. In this connection, reference must be made to certain forms of congenital scoliosis which show maldevelopment of vertebral bodies, fusion of the ribs, and not infrequently also elevation of the scapula at the side of the convexity. These defects in the normal development of ribs and vertebræ point to a more elementary developmental error than would be represented by a mere failure of the scapula to descend. Of the muscular changes which are seen in the congenital elevation of the scapula, the most remarkable are those of the trapezius muscle, a complete absence of which has been reported by Kirmisson. Cases have also been reported with absence of the sternocleidomastoid muscle and defects of the pectorales muscles. The serratus magnus, the levator anguli, the latissimus dorsi, and the rhomboid muscles are also occasionally involved. Deformities of the humerus and of the clavicle have also been reported.

The clinical symptoms of the deformity depend largely upon the pathological position of the scapula. The coexistence of scoliosis of varying degree has already been mentioned. The curvature is usually situated in the lower cervical and upper dorsal spine producing a compensatory curve in the dorsal and lumbar sections. Only in a certain percentage of cases scoliosis exists. It is probably an incidental deformity and caused by the same general arrest of development. In the same way, too, torticollis is seen in a certain percentage of cases but there is no direct connection between its existence and the high position of the scapula. In not less than half of the cases, the function of the shoulder as regards motion is practically unimpaired. Where an impairment of function exists, it is essentially in the direction of abduction of the arm, which cannot be brought beyond the horizontal. Since abduction in the scapulohumeral joint is only possible to the horizontal, and since beyond this plane abduction is dependent upon rotation of the scapula itself, this restriction of abduction can be readily understood. Even in cases of bilateral deformity the range of motion of the arm in abduction is not necessarily restricted to a marked degree. In these bilateral cases the shoulders appear as being forcibly drawn up, the neck is very short and the normal contours of the neck and shoulder line are changed from a gentle slope as seen in normal posture, to a more or less acute angle.

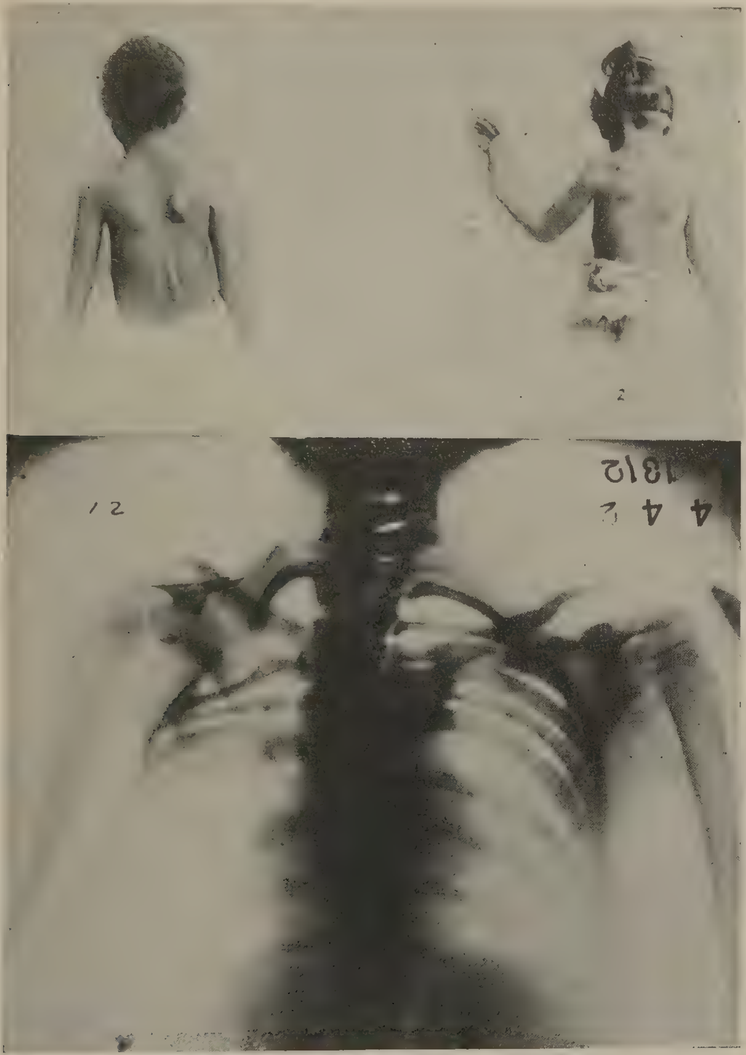

FIG. 211.—Congenital elevation of scapula.

Treatment.—In milder cases gymnastic exercises and stretching of the contracted muscle may be carried out with success. In cases in which conservative methods do not produce results and where there is enough interference with the function to make radical measures worth while, several operative methods may be carried out.

1. The technique used by Wilson and La Rue is as follows: An incision is made over the vertebro-scapular articulation, the bone bridge is resected, and the scapula is thereby freed. In order to reach it, the rhomboid and trapezius muscles often have to be dissected from their places of insertion. In a number of cases this will make the scapula freely movable so that it then may be held in proper position. In some cases, however, the hooked superior angle of the scapula must also be resected. After division of the lower portion of the trapezius which often consists of dense fibrous bands only, and of the rhomboid and levator anguli, the cartilaginous bands attached to the upper inner angle of the scapula are then approached and removed. Then the shoulder blade appears completely mobilized and may now be depressed downward.

2. Mobilization of the Scapula.—In the case of B. Gill, the scapula could not be brought down to normal position even after complete removal of these articulations, until the trapezius was separated from the posterior portions of the spine of the scapula and the two rhomboids as well as the levator anguli were also detached. In addition, the supraspinous muscle had to be freed from its posterior attachment, and the hooked upper and outer angle of the scapula had to be resected together with the entire upper portion of the bone as far as the suprascapular notch.

These reports show that operative correction of the deformity may often be extremely difficult, and calls for very extensive resection of both bone and muscles. Naturally, such extensive interference is hardly justified unless the deformity is accompanied by considerable functional impairment of the shoulder.

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SHOULDER PARALYSIS

Muscle Dynamics.—The management of paralysis of shoulder muscles irrespective of the underlying cause is so intimately related to questions of muscle dynamics that a short discussion of the latter should precede the description of clinical syndromes.

The movement of the scapula is either translatory, in which case all points move parallel, or rotatory around a central axis. There is a great need for unusual mobility of the shoulder blade because of the fact that it follows closely all movements of the arm, aligning itself with the long axis of the humerus. The reason for this is essentially dynamic, *viz.*, to impart the impetus of motion freely from the shoulder blade to the entire extremity. It requires an unusual amount of musculature to take care of motion in all directions in which the extremity itself may be moved. In addition to this dynamic requirement, there is also the ever-present necessity of stabilization of the shoulder blade to be accomplished by muscle action also, since the only bony connection of the scapula is its syndesmosis with the clavicle. Rotatory motion is carried out mainly by the trapezius and serratus magnus, in one, and by the rhomboids and levator anguli muscles in the opposite direction.

Paralysis of the Serratus Magnus.—The paralysis of the serratus magnus muscle is attended by a disability of the shoulder joint quite characteristic for the action of this muscle. Since the serratus magnus holds the shoulder blade tightly to the posterior chest wall, its deficiency results in the receding of the vertebral border from the chest wall which gives the shoulder a winged-like appearance (Winged Shoulder). Since, furthermore, the serratus magnus muscle is an important rotator of the scapula synergistic with the trapezius in the act of abducting the arm from the horizontal to the perpendicular line, the paralysis of this muscle also causes inability to abduct the arm above the horizontal.

Isolated paralysis of the serratus magnus muscle is rare. It may be due in exceptional cases to infantile paralysis or more often to peripheral paralysis following injury to the long thoracic nerve, for instance, after neck operations. The characteristic disability of the arm offers as a rule little difficulty for the diagnosis. The writer has observed two cases of this kind. One case was that of a girl eight years old who had an attack of infantile paralysis two years previously. The disease involved the left shoulder only. She was able to elevate the arm up to the horizontal but it was difficult to get beyond. When elevating the arm in a forward plane, the scapula was seen to rotate about the vertical axis, the inner border losing contact with the thorax. In the other case, the patient was 18 years old and complained of deficiency of the right shoulder for the last six years, which had reached a stationary point two years ago. The difficulty appeared in trying to raise the arm above the horizontal line which was possible only by a sort of swinging motion. When this was done the shoulder blade seemed to rotate about a vertical axis so that its median border became separated from the ribs and come strongly into relief. In this case, the cause of the isolated paralysis of the serratus magnus muscle could not be determined.

Treatment.—Conservative treatment consists in application of an appliance in order to hold the shoulder in place. Such appliances have been described by several authors, but, as a whole, the results were not satisfactory.

For the operative treatment, several methods have been advised. The first to operate on the winged scapula was Eiselsberg in 1878. His method consisted in suturing together the vertebral borders of the shoulder blades in midline; and, in a second step, the superior inner angles were also united. This procedure, however, severely handicaps the free mobility of the shoulder and is therefore open to grave objections.

A better procedure is probably that of Duval reported in 1903, in which the scapula is fixed to the posterior chest wall by a periosteum-muscle flap in a position which corresponds to that of extreme abduction, *viz.*, with the vertebral border slanting downward and outward. Finally, a nerve plasty has been tried and reported by Skillern. He used the short subscapular nerve for the implantation into the long thoracic. Inasmuch as no end results were reported, little can be said about this method.

Deltoid Paralysis.—Deltoid paralysis is the most frequent paralysis among the scapulo-humeral group of muscles. The deltoid is the only abductor of the arm capable of doing any substantial amount of work. It can be divided into three portions: The anterior and posterior portions abduct the arm as well as carry it forward or backward respectively, while the middle portion is a pure abductor. It is the function of the deltoid muscle to elevate the arm from the side of the body to an angle varying in different planes. In forward abduction this angle is about 130°; in abduction to the side, or in the frontal plane, the range is 90°; while in abduction in the oblique posterior plane the range is about 45°. Beyond these angles further abduction is taken care of by rotation of the shoulder blade which is carried out by the scapulo-thoracic muscles, especially the trapezius and serratus magnus.

Deltoid paralysis is by far the most frequent paralysis of the upper extremity following anterior poliomyelitis. Among 883 cases of infantile paralysis of the writer's series, 70 involved the upper extremity, and of these, not less than 56 cases involved the shoulder joint with paralysis of the deltoid muscle.

Treatment.—The primary indication in all cases of deltoid palsies following infantile paralysis of more or less recent date is the relaxation of this muscle by means of splinting in the hope of facilitating the spontaneous recurrence of power of this muscle during the period in which the natural repair of paralyzed muscles is known to take place. The chances of spontaneous recovery of this muscle will be duly appreciated when one considers that permanent and complete paralysis of the muscle is by far in the minority as compared with temporary and partial paralysis. Of 16 more or less recent cases in which the period elapsed since onset varied from eight weeks to two years, 12 were treated by splinting. A complete recovery of the deltoid muscle was obtained in three cases and a decided improvement in the power of this muscle was obtained in four more cases, making a total of seven cases recovered or markedly improved though in one case such improvement was obtained as late as two years after the onset of the paralysis. This considerable percentage of recovery shows the correctness of the indication of conservative splinting. This is combined with massage, active and passive exercises, the latter being carefully controlled so that no fatigue or exhaustion of the recovering muscle may occur. It is especially necessary to retain the patient's arm in the position of right angle abduction without interruption and when the splint is removed for the purpose of treatment, it must be done with special precautions and the arm held abducted so that the deltoid muscle never ceases to be relaxed.

Operative Treatment.—Operative interference is justified only if the deltoid muscle fails to present any signs of activity after the lapse of a reasonable time; if it shows no electro-diagnostic response, and if its degeneration and atrophy is so evident as to put recovery beyond all expectation.

It is also necessary to ascertain in case of deltoid loss whether or not abduction of the arm may be possible by substitutionary motion to a degree sufficient for average function of the extremity. Some patients learn to use the contraction of the biceps and triceps muscles as a means to stabilize the humerus against the glenoid fossa, and they subsequently manage to abduct the arm by means of the rotators of the shoulder blade. However, in the majority of cases, the substitutionary motion is inadequate and will not stand comparison with the results of operative interference. If then on the grounds of failure to obtain recovery of the muscle itself or to develop substitutionary motion to a satisfactory degree, operative methods are decided upon, the choice is to be made between tendon transplantation and fixation, or arthrodesis of the shoulder.

Regarding tendon transplantation, it cannot be denied that there is an abundance of muscle material in the neighborhood which might be used for transplantation purposes. Winiwarter used the clavicular fibers of the pectoralis major. Hoffa used the trapezius and pectoralis major muscles for implantation upon the paralyzed deltoid. Good functional results following these methods have occasionally been reported. The technique of these operations is not without difficulty, especially in view of the fact that the thoracic nerves and vessels reach the pectoralis major from its under surface and their integrity is absolutely essential for the success of the operation, a fact which interferes greatly with the free detachment of the flap. There are also great difficulties in bringing down a flap of the trapezius muscle to a point of the humerus where its insertion will be of mechanical advantage.

For these reasons, it is best to return to methods of fixation of the shoulder joint rather than to those of tendon transference. Bartow and Plummer advocated in 1913 the use of silk ligaments inserted into acromion and head of the humerus in order to hold the joint in position of abduction. The technique as described by these authors is simple, and the operation has certainly some merits, especially in young children in whom one might hesitate to perform more extensive operations upon the joint, but late end results are not at hand, and the method does not seem to warrant expectations of permanent results.

The only method which can be relied upon to give permanent results is the arthrodesis of the shoulder joint, a method advocated even by Lorenz who is generally in opposition to arthrodesis operations. The first arthrodesis was performed by Albert in 1879.

Bradford, Whitman, and Sir Robert Jones are advocates of this arthrodesis. Vulpius in 1912 reported 12 cases of flail shoulders treated by arthrodesis in which he obtained an average amount of forward flexion and abduction of 75°, and an average amount of straight lateral abduction of 60°.

Age.—It is not necessary to wait until the fourteenth or eighteenth year of life as Lange advocates, but the arthrodesis may be safely performed in young children. Sir Robert Jones advocates arthrodesis as early as at six to eight years of age. When done at that age a greater amount of bone must be removed from the articular ends in order to produce ankylosis, but this is outweighed by the advantage of procuring early functional development of the extremity. While bony ankylosis of the joint is always the more desirable outcome, fibrous ankylosis obtained in smaller children gives very acceptable results.

Angle.—As to the angle at which fusion should be accomplished, different suggestions are being made by different observers and operators. For children, the right angle abduction is undoubtedly the better position. In adults, however, an angle of 60 to 70° is preferable.

Technique.—The patient lies on his side with the shoulder supported by sand-bags. During the operation a downward pull upon the shoulder is made by one of the assistants standing on the side opposite the operator. An incision is made from the spine of the scapula forward encircling the acromion and ending anteriorly over the coracoid process. After skin and fat are severed, a flap is prepared and turned upward. The flap should reach upward high enough to expose the edge of the acromion for a distance of 1 to 1½ in. One then proceeds to divide bluntly the fibers of the deltoid at places corresponding to the anterior and posterior border of the middle portion of this muscle. The separation is carried upward to the acromion and downward to the insertion of the deltoid muscle. Then a grooved director is pushed under the muscle and the acromion is next incised transversely ¾ to 1¼ in. from its edge, and then cut through with an osteotome. This gives a flap consisting of the middle portion of the deltoid or the curtain of fibrous tissue representing it, to which is attached the severed end of the acromion. This bone muscle flap is turned downward. Now the entire anterior surface of the shoulder joint capsule is in view. The capsule is opened by a longitudinal incision, the edges of the incision being carefully secured by a forceps and widely retracted. As the arm is then pulled downward the glenoid fossa can be seen entirely, and also most of the head of the humerus. The long tendon of the biceps is in front of the incision passing through the joint. Next the cartilage covering of the glenoid fossa is completely removed. This is followed

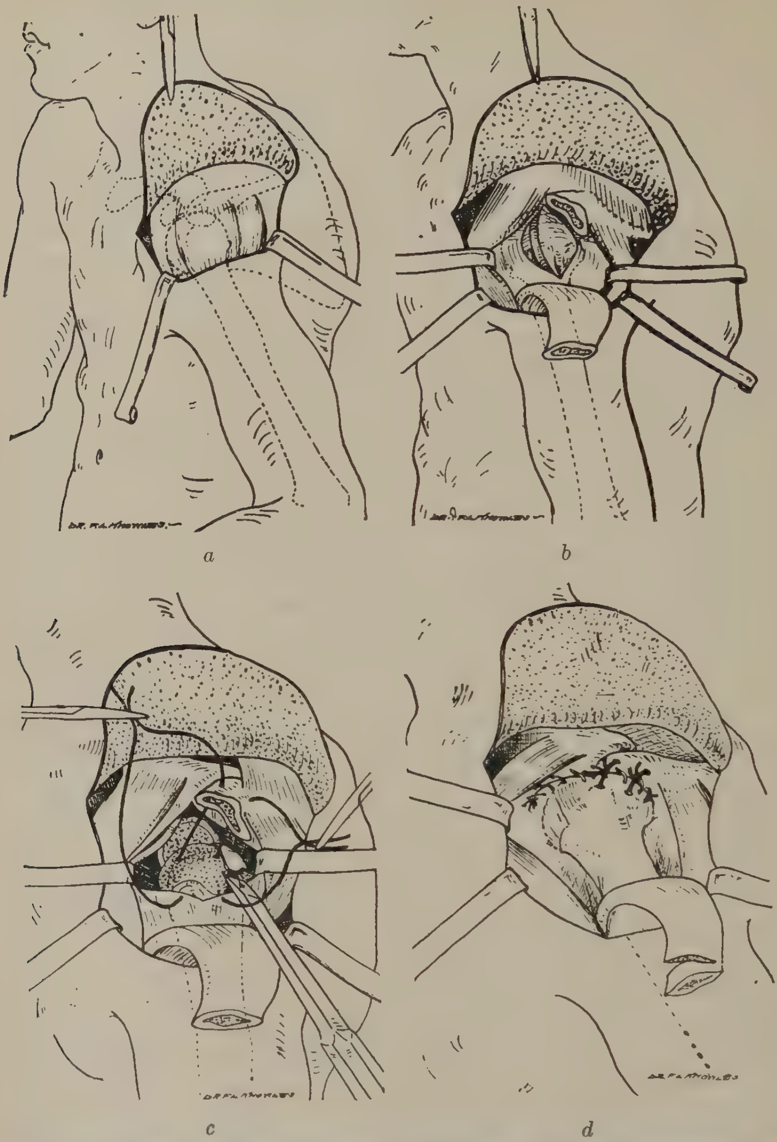

FIG. 212.—Technique of arthrodesis. *a*, Incision. *b*, Deltoid flap turned down. *c*, Removal of cartilage; placing of drill hole sutures. *d*, Suture of capsule; tying of drill hole sutures.

by removal of all the cartilage from the head of the humerus, and to this end the head may be pushed through the incision in the capsule and out through the wound. Then heavy catgut No. 4 is placed through drill-holes made in the acromion and the head Two of these sutures are passed, one more anteriorly and one more posteriorly; but they are left untied as yet. The capsule is now closed by suturing a longitudinal slit transversely by which maneuver a reefing and tightening of the anterior capsule is effected. Then the arm is placed in abduction and is maintained in this position throughout the remainder of the operation and during application of the cast. Now, the heavy drill-hole sutures are tied. A muscle flap consisting of the acromion and deltoid is now carried backward after the adherent piece of acromion has been resected. This flap is fastened to the spine of the scapula. It thus serves as a posterior cushion counteracting the tendency of the humerus toward posterior displacement. The wound is then closed in layers and a plaster-of-paris cast is applied reaching from the shoulders

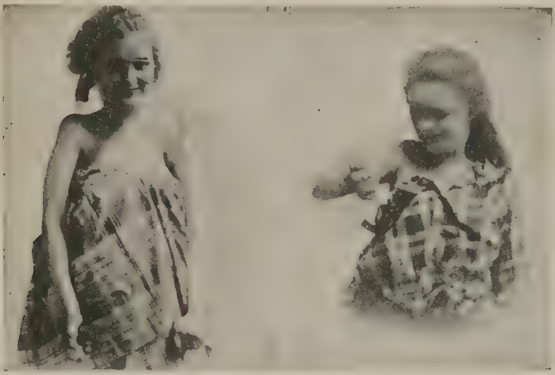
a *b*

Fig. 213.—*a*, Paralytic flail shoulder. *b*, After arthrodesis.

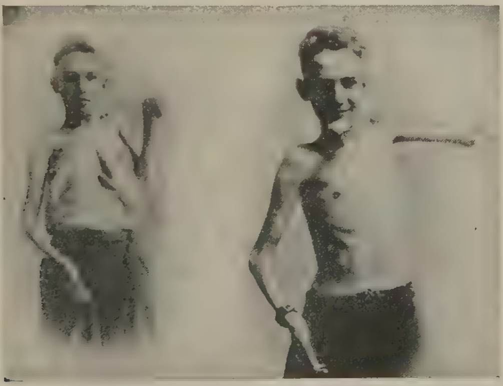
a *b*

Fig. 214.—*a*, Paralytic flail shoulder. *b*, After arthrodesis.

to the hips and encircling both arm and forearm. The position is abduction and slight forward flexion with inward rotation of the arm and flexion at the elbow.

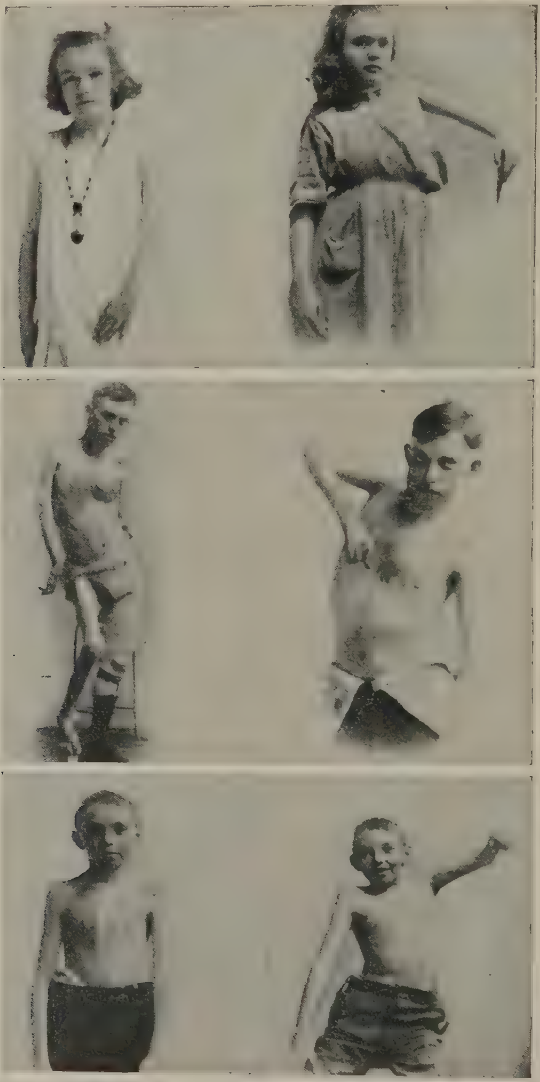

FIG. 215.

The cast is left in place for two or three months, the wound being taken care of through a window. At the end of this time the cast is removed and supplanted by a platform splint already at hand. From then on, the mechanical after-treatment begins. The after-treatment consists in massage of the muscles of the shoulder, especially of the trapezius; in massage of the arm and forearm muscles, and in active exercises. The exercises also are carried out under strict supervision, and active elevation must keep step with the development of the shoulder muscles and over-exercising must be carefully avoided. Not until one is sure of the power of active abduction of the arm may the angle of the splint be decreased. This may take three months or more after removal of the cast. After the splint has been gradually lowered, it may finally be discarded, but the patient should be directed to keep up his active exercises.

One point of the after-treatment must be strongly emphasized. This is that the unusual position of the arm and especially the difficulties of bringing the arm to the body-line as they exist during the first year after operation, are liable to produce curvature of the spine or to accentuate an already existing paralytic curve. This must be guarded against by vigorous scoliosis exercises and by the application of a brace or corset which insures the support of the body without interfering with the activity of the shoulder.

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CHAPTER CXVI

THE ELBOW JOINT

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THE FLAIL ELBOW

The flail elbow joint is produced most frequently by the loss of muscle power due to infantile paralysis, by injuries to the brachial plexus, often following gunshot wounds, or by isolated injuries of the musculocutaneous or musculospiral nerves.

Especially paralysis of the musculospiral nerve is a frequent complication of fracture of the humerus. A flail elbow joint may also be the result of extensive destruction of the articular ends such as follows extensive wounds of the elbow with involvement of the bone.

The paralytic flail elbow in which the joint bodies are intact, such as seen in infantile paralysis or peripheral paralysis, calls for different methods of treatment than those applied to flailness of the elbow joint as such. It is here the problem of reëstablishment of motion of the elbow joint while in the other instance it is that of stabilization of the joint.

In establishing motion of the elbow joint one should be cognizant of the fact that a close dynamic relationship exists between elbow and shoulder. By abduction of the humerus, the sweep of the motion for forearm, hand, the fingers is greatly increased, and since in this horizontal abduction, forearm and hand exert very little gravital force, it is obvious that flexion and extension movement of the elbow as well as pro- and supination can in this position be carried out with the least display of force. Under such favorable conditions it is possible to bring into use for elbow motion substitutionary muscles which otherwise would have very little effect upon motion in the elbow.

If the flexors or extensors of the elbow are weakened as in infantile paralysis, the primary indication again is appropriate splinting which must be carried out in such a way that the joint is held in horizontal position. The splint allows the arm to rest horizontally, the shoulder being in abduction and the forearm pointing forward in a position of right-angle flexion with the arm so that the weakened flexor muscles of this joint may be duly relaxed. A large number of cases, however, will not regain muscle power in due time, and for these cases operative procedures may be applied.

A diamond-shape piece of skin may be removed out of the fold of the elbow according to the method of Robert Jones, and the wound sewed together holding the elbow in position of flexion.

Arthrodesis of the elbow may also be considered in older patients, but the procedure is complicated and not easily adaptable to children under fourteen years.

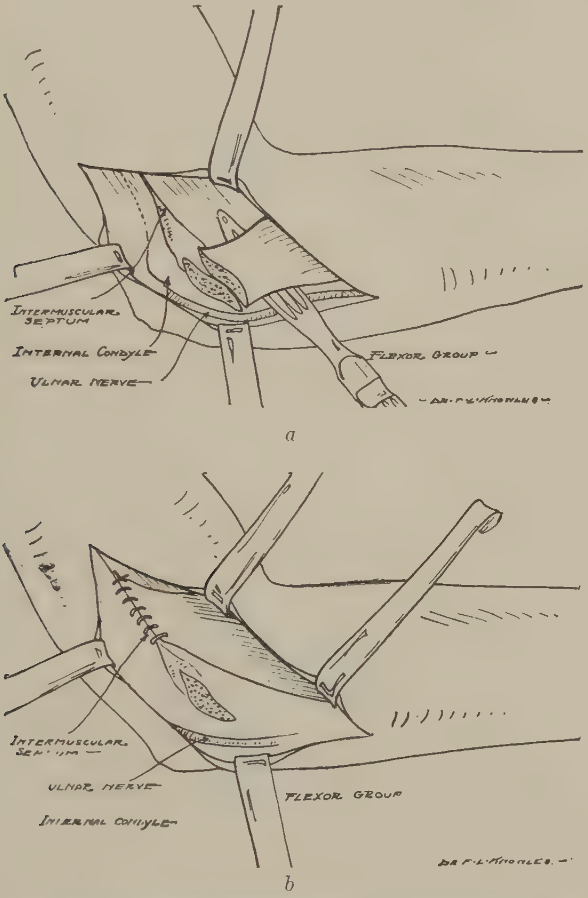

Fig. 216.—Technique of muscle transposition. *a*, Dissection of muscle flap. *b*, Attachment to intermuscular septum higher up.

The writer uses a method of muscle transposition in which he makes use of the slight flexion power upon the elbow of certain flexors of the forearm and hand, *viz.*, the flexor carpi radialis, flexor carpi ulnaris, the pronator teres, and the palmaris longus. The method consists essentially in the transposing of the common origin of these muscles at the inner epicondyle of the humerus to a place higher up upon the humerus. This arrangement changes the dynamic conditions of these muscles in such a way that their flexion action upon the elbow becomes greatly increased.

If to this is added the ability to elevate the arm to the horizontal as is implied by the arthrodesis operation of the shoulder previously described, then both arm and forearm play, as it were, in a horizontal plane, and without the strain of gravity, and the newly created flexors of the elbow find their power increased to a degree of practical value. Indeed in most cases of this kind, the shoulder is involved as well as the elbow, and the result of the combined surgical interference upon elbow and shoulder is a certain degree of usefulness for the entire flail extremity. The active motive power for the elbow is furnished by the flexor muscles of wrist and fingers, and for the shoulder joint by the muscles of the scapula.

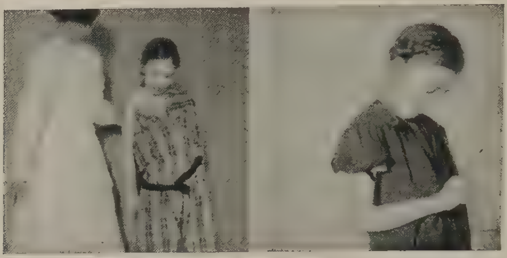
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FIG. 217.—*a*, Paralytic flail elbow. *b*, After muscle transposition.

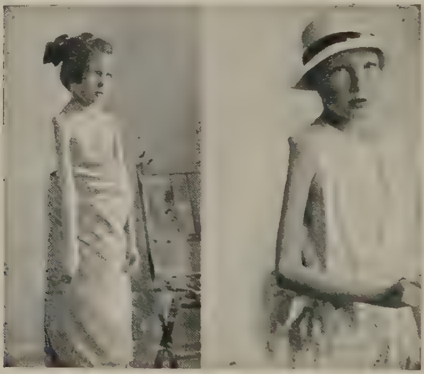
a b

FIG. 218.—*a*, Paralytic flail elbow. *b*, After muscle transposition.

In the writer's series, 25 cases were operated in the manner described, and the results on the whole were quite gratifying.

CONGENITAL DEFORMITIES OF THE ELBOW

Radio-ulnar Synostosis.—The seat of congenital synostosis of the forearm bones is usually at their upper end, the union consisting in a broad, bony bridge which unites the corresponding borders of radius and ulna. The radio-ulnar synostosis is as a rule an absolute obstacle to pro- and supination movement. The forearm is held in extreme pronation, and neither passive nor active supination movements are possible. One case of this kind has been observed by the author. A radio-ulnar synostosis existed in the right forearm which was in position of extreme pronation with supination entirely arrested while flexion and extension of the elbow were free. The *x*-ray showed a synostosis of the proximal radio-ulnar joint.

The only possible treatment of cases of this type is the operative resection of the entire synostotic bridge.

This procedure involves the resection of more or less of the upper end of the radius according to the width of the bridge. Resection of the bone bridge alone either with interposing of a muscle flap does not seem to give lasting results unless the upper end of the radius carrying the bone bridge is also resected.

Congenital Synostosis of the Humerus with the Forearm Bones.—Anomalies of this type show a more or less complete fusion between the humerus and either radius or ulna. In the majority of cases it is the ulna which appears fused with the humerus. Most of these cases are also afflicted with other congenital deformities, especially of the forearm and hand.

The writer has observed two cases of congenital synostosis of the elbow joint which were both associated with other deformities. In one case, a boy of four, there existed an ectrodactyly with complete synostosis of the radius of the humerus on the left side, and a complete synostosis of the ulna with the humerus on the right side. The musculature of the arm and forearm was very much under-developed and badly disarranged so that clinically a differentiation between flexors and extensors of the elbow or pro- and supinators could not be made. The second case was a child six months old with cutaneous syndactyly and symphalangism in which there also existed complete fusion of the radius with the ulna on the right side while the left elbow was entirely free.

Congenital Dislocation of the Radius.—The deformity is rather rare though W. E. Blodgett in 1906 was able to report on 51 cases including two of his own.

The cases occurred bilateral or unilateral, nearly two-thirds of the cases being found in male children. The commonest direction is backward dislocation, forward dislocation being nearly as frequent while outward dislocation of the radius is comparatively rare. In many cases, the proximal end of the radius appears lengthened, and in one-third of the cases the condition was complicated by radio-ulnar synostosis. A case of this kind was observed by the writer. It was a

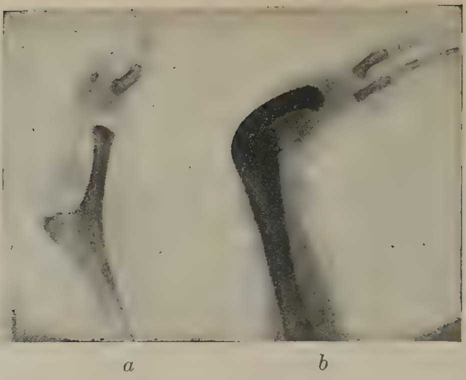

Fig. 219.—*a*, Synostosis of humerus and ulna. *b*, Synostosis of humerus and radius.

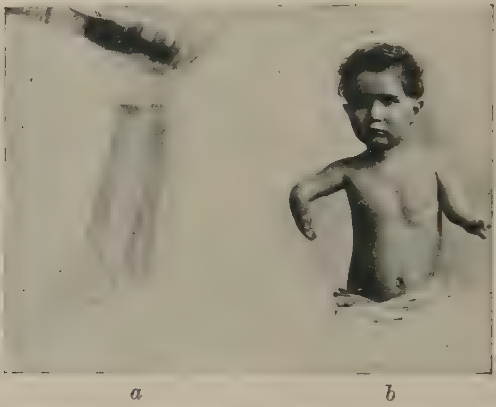

Fig. 220.—*a*, Synostosis of humerus and radius. *b*, Case of Fig. 219 *a* and *b*.

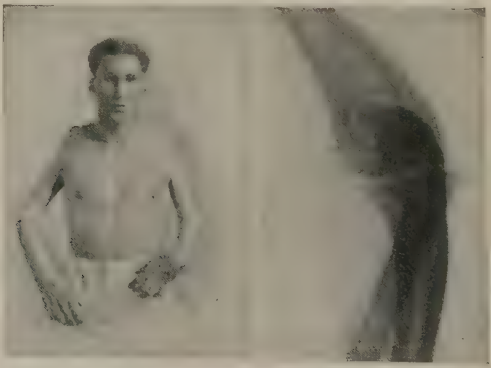

Fig. 221.—Congenital ankylosis of elbows.

patient who had obstetrical paralysis of the whole-arm type. The radius was displaced forward and could be forced back in place but could not be held there. The treatment in this case consisted in cutting down upon the radial head and in reconstructing the annular ligament, splinting the arm in acute flexion. The immediate result of the operation was that the radius was held in place and flexion became free. The late result could not be observed as the patient was lost sight of.

Where considerable restriction to flexion and extension exists, excision of the head of the radius is indicated. Cases with free flexion and extension but permanent pronation due to bone fusion of the forearm bones require the resection of the bone bridge together with the proximal end of the radius as already indicated above.

TRAUMATIC DEFORMITIES OF THE ELBOW

Cubitus Valgus.—Cubitus valgus deformity of the elbow is often seen as a result of a fracture involving the external condyle. The normal carrying angle of the elbow is 10 to 15° in men and about 20° in women. In the latter, a considerable increase of the carrying angle takes place during puberty owing to the increasing width of the pelvis. In the pathological cubitus valgus the carrying angle is increased. The most common cause of this deformity is fracture of the external condyle of the humerus. Cubitus valgus deformity is also known to occur from occupational strain such as heavy lifting, in which case we must assume that there is a certain natural predisposition to this deformity consisting in a laxity of the lateral ligaments.

Cubitus valgus has also been observed following peripheral paralysis of the median and ulnar nerves. In this instance the deformity is due to a disturbance of the muscle equilibrium occasioned by the loss of the flexor muscles arising from the inner condyle. It seems that when the median nerve alone is paralyzed, the surviving flexor carpi ulnaris is sufficient to counterbalance the muscles coming from the outer condyle of the humerus but that in paralysis of both nerves the action of the extensor group arising from the outer condyle forces an unopposed abduction of the forearm resulting in cubitus valgus deformity. Treatment is indicated in advanced degrees of the deformity or where impairment of function exists. The usual method is a supra-condylar osteotomy of the humerus followed by correction of the deformity and immobilization in plaster until the solid bony union has occurred.

The Traumatic Cubitus Varus Deformity.—Cubitus varus deformity also follows frequently fracture of the elbow joint. The most common type of fracture which leads to this deformity is the fracture of the internal condyle or the intercondyloid fracture of the humerus. It is, however, not so much the lateral deviation in the elbow joint as the displacement of the fragments either in front or behind the joint-line which determines the degree of disability. Of five cases of cubitus varus which were observed by the writer, two followed fractures of the internal condyle, two fractures of the outer condyle, and one an intercondyloid fracture of the humerus. In many cases the deformity is associated with more or less complete ankylosis of the elbow and considerable impairment of function.

In cases in which the deformity is due to intra-articular fractures (and these form the majority), it is necessary to distinguish between intra- and extra-articular obstacles to free motion. Poor function is often due to extra-articular adhesions while the articular ends themselves may be more or less intact showing a normal, smooth cartilage covering. The displaced fragments often act by themselves as mechanical obstacles.

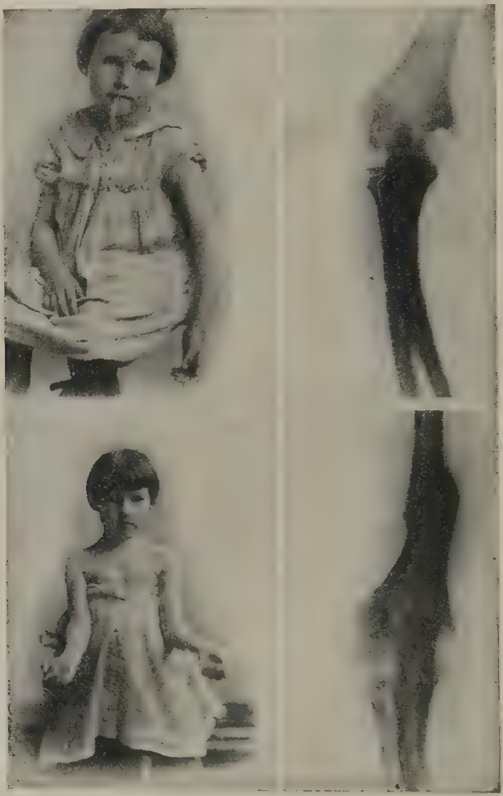

FIG. 222.—Traumatic cubitus varus deformity of elbow: Before and after operation.

Treatment.—The distinction between deformity as such and deformity combined with partial or total ankylosis determines the treatment to be adopted. If deformity alone is present the treatment consists in osteotomy of the humerus directly above the elbow and in correction of the deformity along similar lines as are followed in cubitus valgus deformity. It is not difficult in these cases to restore the normal carrying angle. Still, unless the deformity is quite pronounced, operation is not warranted unless functional impairment exists.

In the latter event, the treatment of the deformity itself is secondary and that of the ankylosis of primary importance. Extra-articular adhesions may be treated by manipulations, stretching, forcible motion, etc. In true ankylosis, however, definite operative methods are indicated among which the arthroplasty occupies first place. The indications and the operative technique of this method will be discussed in a later chapter.

Elbow Strain.—Elbow strain may be caused by sprain of the extensor muscles of the forearm at their point of origin at the external condyle of the humerus or in the immediate neighborhood. These strains form a distinct clinical entity which by many is explained as injury to the lateral ligament of the elbow joint which arises from the external condyle of the humerus and spreads fan-shaped downward serving as point of insertion for the extensor muscles of the forearm. When this ligament is sprained or torn, direct pressure upon the external condyle of the humerus elicits pain. Lately, it has also been pointed out that a traumatic synovitis may occur in the various bursæ underlying the forearm muscles. One type described by Osgood as tennis elbow is due to inflammation of the bursæ underneath the extensor carpi radialis. It is of importance that sprains of the elbow do not always come about by a single direct or indirect trauma but they often occur as occupational sprains caused by continuous and gradual exertion of the extensor muscles. Such conditions have been described occasionally. Remak and Bernhardt classify them as joint neuralgias while Oppenheim considers them as occupational neuroses.

The treatment of sprains of the elbow consists in fixation in splint or cast followed by massage, and later by active and passive motion.

Traumatic Ankylosis of the Elbow.—Fracture deformities of the elbow which result in ankylosis are not uncommon. Usually it is the intra- or supracondyloid fracture of the lower end of the humerus which produces this disability. Very often there is a displacement of a fragment either in front or behind the elbow joint which forms a mechanical obstacle to flexion or extension. But these bony obstacles are by no means the only difficulties nor are they in the majority of cases the principal sources of functional impairment. Quite regularly one finds very extensive and very strong extra-articular adhesions, largely produced by the organization of the large hematoma which accompanies the injuries to the soft structures surrounding the joint. These extra-articular adhesions play a large, if not the larger part in restricting the motion of the elbow, and their existence must be duly considered when plans are laid for appropriate treatment.

Treatment.—Peri- or extra-articular adhesions offer a much greater possibility for mechanical treatment, *i.e.*, forcible stretching, passive motion, and mobilization under anesthesia than do intra-articular lesions. The treatment must be carried out for a considerable length of time, half a year to one year being by no means an unusual duration. At the same time, proper judgment and caution must be exercised because of the reactions which forcible manipulation may set up in the soft structures. If the treatment is too vigorous, reflex muscle contractures occur which can be overcome only by persistent, judicious, and cautious treatment. Often a relaxation may be obtained only by a short period of fixation. Upon removal of the mobilizing cast one will then find the joint much freer and passive manipulation carried out subsequently will then produce much better results.

When disability is due to displaced fragments in front or behind the elbow joint, their operative removal is indicated. They may be dissected without materially interfering with the integrity of the joint itself. When the operative wound is closed, the post-operative mechanical treatment should begin and should be carried out consistently for many months. No fixed rule can be set for the manner in which the mechanical treatment is to be applied. According to the state of reaction, periods of fixation may have to follow periods of mobilization, and the indications vary so frequently in individual cases that only a continuous supervision and the application of sound judgment will yield the desired results.

One of the cases observed by the author was a boy 16 years old who had sustained a fracture of the left elbow five years previously. When seen he was unable to extend the elbow. The examination showed that the lateral condyle was detached from the outer side of the humerus to a point in front of the elbow where it could be felt under the skin. The extension of the arm was checked at 150°, the flexion at 80°. The detached piece of the external condyle was resected and removed, and this was followed by an extensive resection of extra-articular adhesions which were found in the fold of the elbow joint. The arm was then placed in plaster for two weeks after which time the mechanical aftertreatment was started. In due time the elbow attained a normal range of motion.

In another case, a boy of 13, the elbow had been fractured half a year ago, the examination showing that the flexion of the elbow was checked at right angles. The *x*-ray picture showed a fracture of the lower end of the humerus, a fragment being displaced forward. Following a resection of the fragment a series of casts was applied and this was followed by mechanical aftertreatment with the result that the range of motion finally reached from 40° to 145°, equalling 105°.

Undoubtedly, one is liable by too vigorous or too early mechanical aftertreatment to lose some of the motion which is regained at operation. For this, the re-formation of extra-articular adhesions is responsible. This situation can be met by the adoption of more conservative means, and, especially, by the prolongation of the fixation period which follows the operation. On the other hand, there are undoubtedly many cases in which the joint itself is sufficiently damaged so that the resection of the bone fragment alone and the removal of extra-articular adhesions is not sufficient to secure a satisfactory range of motion. In this group of cases, arthroplasty should be employed.

Arthroplasty of the Elbow Joint.—With interposition of soft tissue between the joint ends, the technique of arthroplasty has made a tremendous stride forward. The interposition method was introduced in this country by Murphy in 1905. In the upper extremity, the elbow joint lends itself most favorably to the performance of arthroplasty. According to Henderson's table covering 126 cases of arthroplasty of the elbow operated by various surgeons, the results were good in 76 per cent. fair in 16 per cent. and poor only in 6 per cent. of the cases. Arthroplasty of the elbow is not applicable to childhood. It should be reserved to the age above 14 to 16 years when the ossification of the articular ends has progressed far enough so that the resection of the bone incident to this operation may not be so extensive as to result in a flail joint and in considerable interference with the growth of the bones. But it must be argued for the elbow as well as for the upper extremity in general that interference with growth does not weigh as heavily as it does in the lower extremity, and that, on the other hand, the advantages of early restoration of function in the upper extremity must be seriously considered.

Albee uses a vertical incision on the posterior surface of the elbow directly over the olecranon (Langenbeck's Incision). The ulnar nerve is displaced and retracted and the olecranon is sawed across and with the adherent triceps tendon is deflected upward. Then follows the separation of the synostosis between humerus and ulna, which is best carried out with an osteotome. After the bones have been cleanly separated, a free fascia flap is inserted, the edges of which are secured to the capsules on all sides. The ulna nerve is then replaced, the olecranon is sutured or pegged to its base, and the wound is closed.

McAusland, who has reported excellent results, uses a U-shaped incision beginning 3 in. above the elbow joint laterally, then passing across and over the olecranon to the inner side of the humerus and from here upward. Here also the ulna nerve is defined and retracted, the olecranon sawed across and deflected upward. A fascia flap is raised from the upper end of the wound, interposed, and the olecranon replaced. This author puts great stress upon the accurate and tight suture of the capsule and the soft structure overlying it in order to obtain a stable joint.

The technique given by Ashhurst affords an excellent approach to the joint. He makes an incision which begins at the external supracondyloid ridge of the humerus about 5 cm. above the joint and which is continued downward to the joint and from here downward and slightly backward on the forearm. The soft parts are cleared away from the humerus, the supinator longus and extensores carpi radialis are placed forward, and the triceps backward until the external condyle is thoroughly exposed. The latter is then detached from the bone with an osteotome. Then the dissection of the joint proceeds forward to the internal condyle of the humerus severing the synostosis between humerus and ulna with a chisel. One is then able to dislocate the entire joint by turning the forearm around the internal lateral ligament as a hinge until forearm and humerus are almost parallel and the fingers point into the axilla. Then the joint ends are made to protrude through the wound and are properly shaped. When this is done, a fascia flap obtained from the superficial surface of the triceps muscle is inserted.

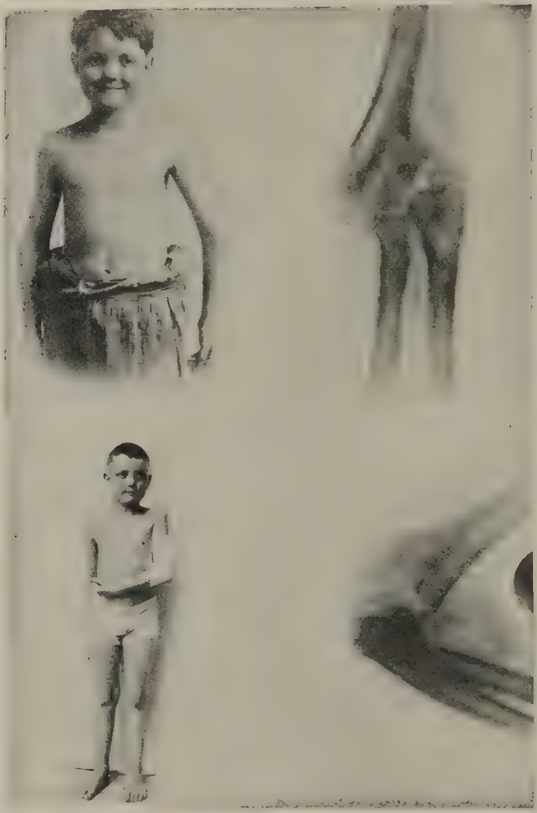

Fig. 223.—Ankylosis of elbow: Before and after arthroplasty.

The writer has employed both McAusland's and Ashhurst's technique in a number of cases with satisfactory results. When doing arthroplasties, one must keep in mind that the prevention of subsequent adhesions depends largely upon the control of post-operative reaction. Allison and Brooks have shown that any inflammatory process within a joint that is sufficiently severe will result in the formation of granulation tissue which destroys cartilage and forms the basis for the union of denuded bone by fibrous bands. If such adhesions are to be prevented it can only be when a cavity is formed between the bone ends by absorption of the tissue used for implantation between the articular ends. But only when the operative reaction is duly restricted such tissue absorption takes place. Formation of abundant granulation tissue is inimical to the formation of a pseudarthrosis or a cavity. Consequently, an operative fixation of the joint following operation

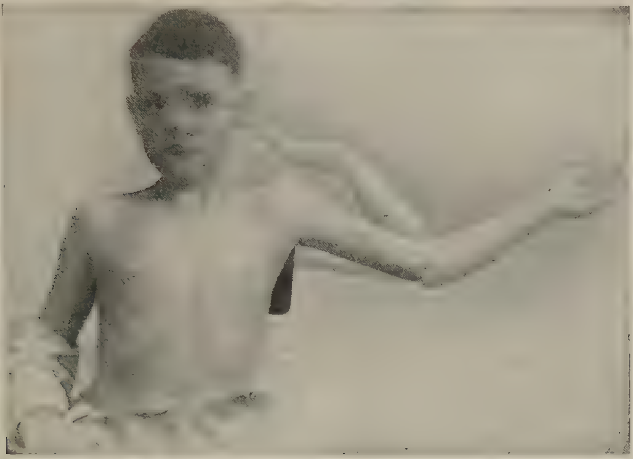

FIG. 224.—Complete ankylosis: Result of arthroplasty.

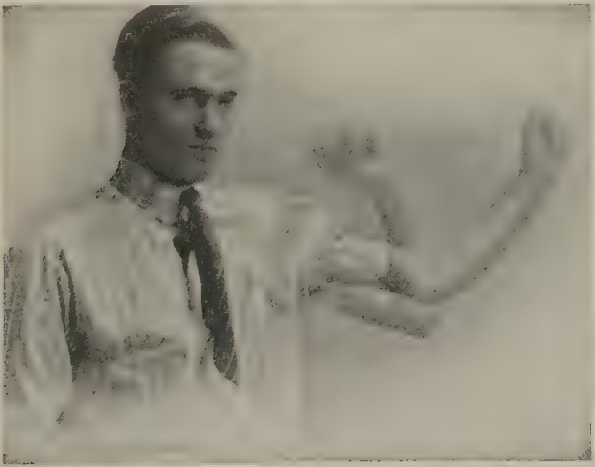

FIG. 225.—Complete ankylosis: Result of arthroplasty.

is necessary from the viewpoint of restricting tissue resection to a minimum.

Among eight cases of arthroplasty of the elbow performed by the writer, a range of motion exceeding 50° was obtained in six, and a range of motion exceeding 70° in four cases, giving 50 per cent. good and 25 per cent. fair results while the result in the remaining two cases was poor.

The aftertreatment of arthroplasty operations is of the greatest importance as without it the operative result even with the best operative technique will be utterly disappointing. We believe that fixation of the joint by cast immediately after the operation should not be less than 10 days or two weeks; after the removal of the cast, the mechanical aftertreatment is cautiously instituted. It should be initiated with gentle massage followed by active motion; passive motion should follow later. Constant supervision of the patient is quite essential inasmuch as indications of the type of aftertreatment vary and occasional reactions make it necessary to modify the treatment.

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CHAPTER CXVII

MALFORMATIONS AND DEFORMITIES OF THE HAND AND FOREARM

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CONGENITAL MALFORMATIONS OF HAND AND FINGERS

Classification.

(*A*) Deformities by developmental suppression, agenesis:
  1. Congenital defect of the forearm bones.
  2. Lobster claw hand.
  3. Ectrodactyly (a-phalangism).
  4. Hemimelia.

(*B*) By developmental arrest:
  1. Syndactyly.
  2. Sym-phalangism.

(*C*) By developmental aberrations:
  1. Polydactyly (some cases).
  2. Hyper-phalangism.

(*D*) Dysplastic conditions:
  1. Chondro-dystrophy.
  2. Brachydactyly (some cases).
  3. Fusion of carpal bones.

(*E*) Polyglandular dystrophy:
  1. Polydactyly (some cases)
  2. Macrodactyly (partial gigantism).
  3. Arachnodactyly.

(*F*) Contractures (neurogenetic or amniotic):
  1. Contracted club hand.
  2. Contracted fingers.
  3. Amniotic contractures (non-symmetrical).

The difficulty of proper classification of a given case lies in the fact that the deformities are often complex and combined with other deformities or malformations. It is not uncommon, for example, to find combinations of polydactyly, syndactyly, ectrodactyly, etc. On the whole, however, the differentiation into deformities by suppression, deformities by arrest, deformities by developmental aberrations, from dysplastic conditions, and from other rarer factors such as neurogenetic deformities, and deformities arising from a polyglandular disturbance, fairly thoroughly covers the field of congenital deformities observed on forearm, hand, and fingers.

Congenital Club Hand.—The true congenital club hand with skeletal involvement is due to suppression of the development of certain bones of hand and forearm. In most cases, it is the first ray including radius, scaphoid, and trapezium, first metacarpal and thumb which have failed to develop. In a minority of cases, the defect involves the ulna and the ulnar ray including the ulnar metacarpals and the ulnar fingers of the hand. The first and larger group of cases is known as radial club hand while the second and smaller group is called ulnar club hand deformity.

Up to 1904, Kirmisson reported 67 cases of congenital club hand, 57 of which showed total and 10 partial bone defect. In 1912, Whitman collected more than 200 cases in the literature. Bilateral club hand is as frequent as unilateral. Complicating deformities such as club foot, congenital deformities of the thorax, fusion of the ribs and many others are quite commonly observed in club hand deformity. The defects do not involve the bone alone but extend also to the musculature. In dissecting a case of total defect of the radius with absence of the thumb, Kirmisson found all the muscles of the thumb as well as the muscles on the radial side of the forearm missing. The remaining muscles are often so badly arranged that their identification becomes a matter of great difficulty.

In cases of ulnar club hand, the skeletal defect involves the ulna, the ulnar carpal bones, usually one or two of the ulnar metacarpals and fingers. In some cases, only the two radial digits remain. The anatomical data regarding ulnar club hand are still more meager than in the case of the radial club hand. Usually the forearm flexors are so badly disarranged that they cannot be identified and the same is true of the extensor muscles of hand and fingers.

The defects in radial and ulnar club hand produce a distinct lateral deviation of the hand, which in the case of the radial club hand is toward the radial side and in the case of the ulnar club hand toward the ulnar side of the forearm. The degree of deviation often exceeds the right angle and in severe cases the hand is completely deflected backward so that its axis becomes almost parallel with that of the forearm bone. Contractures of the skin and of the soft parts on the side of deviation are often so great that the position of the hand cannot even forcibly be corrected.

Another type of congenital club hand shows no skeletal defects but merely contractures of the soft tissues, especially the tendons. According to the position, one may distinguish ulnar, palmar, radio-palmar, radio-dorsal, or ulno-palmar club hands. The etiology of this type of contracted club hands is pretty much undeveloped. The deformity is usually symmetrical and bilateral and for this reason it is hard to apply the theory of amniotic constriction to this type of cases. By some these deformities are considered as club hands in the stricter sense of the word because of their similarity to the ordinary type of congenital club foot. The tendons of the palmar-radial and ulno-palmar sides are contracted and deformities of the finger quite often accompany the deformities of the wrist.

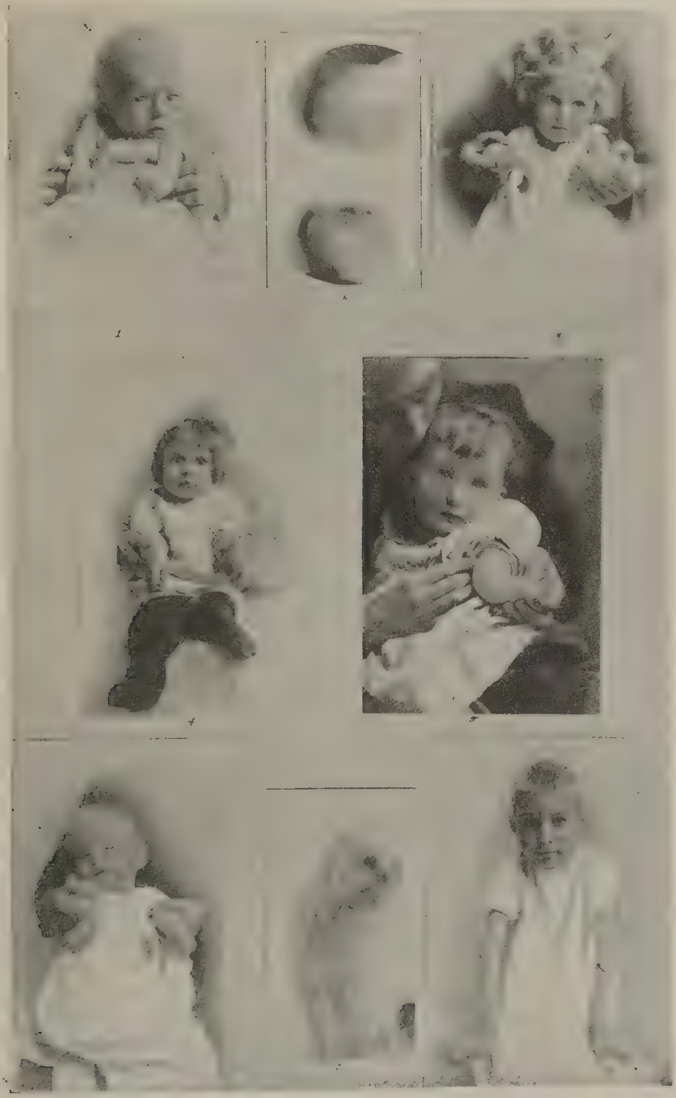

FIG. 226.—Ulnar and radial congenital club hand.

Among a series of 12 cases of congenital club hand which have come under the writer's observation, there were five which showed defects of the forearm bones while in seven no skeletal defects were present. Of the five cases with defects, two were ulnar and three radial club hands. Of the seven cases of contracted club hands, all were of the palmar or palmar-ulnar type, the hand being in position of volar flexion and ulnar abduction.

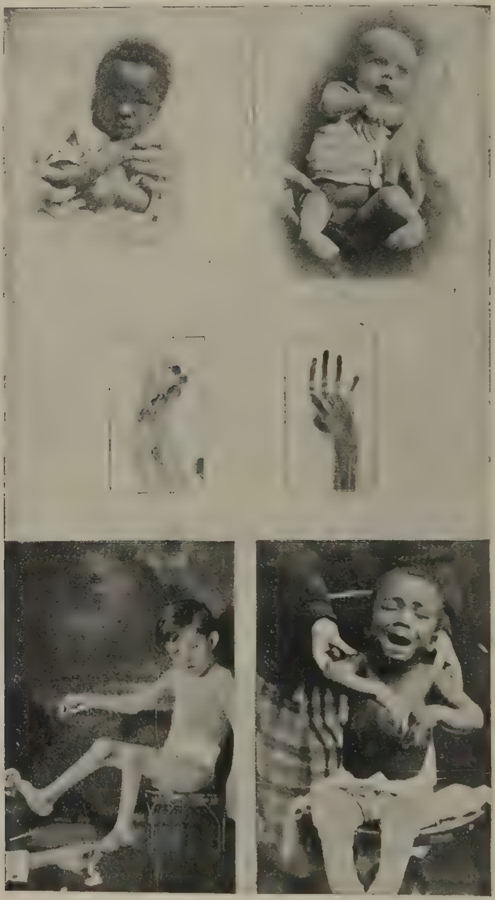

FIG. 227.—Contracted club hands.

Treatment.—In cases of club hand deformity without bone defects the methods of treatment consist in splinting, tenotomies, and tenoplasties. Splinting may be sufficient in mild cases if treated early. It is good practice to begin the splinting at the age of a few weeks. Even in more advanced cases in which operative procedures are necessary, the splinting at very early age is an excellent preparatory method. The more advanced cases require tenoplastic operations to release the contracture. Where a bowing of the radius or ulna exists, this deformity may have to be corrected by osteotomy or by the excision of a wedge-shaped piece of bone in order to straighten the curve in the forearm. The maintainance of the corrected position after operation is a difficult problem of great importance because the success depends upon the persistent and painstaking aftertreatment. Splints must be

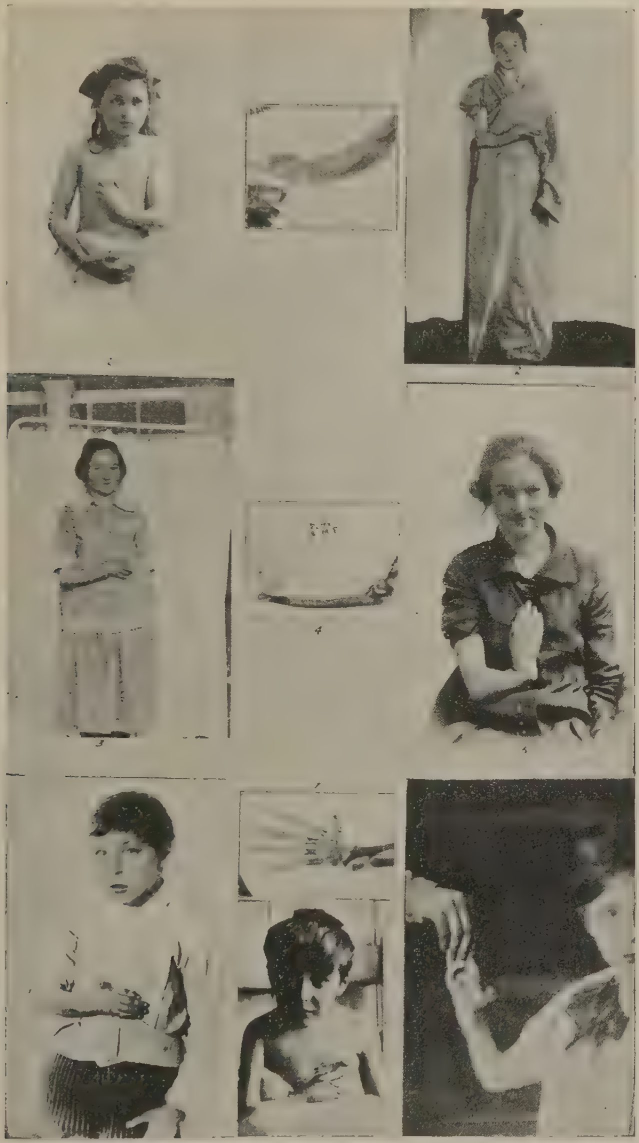
FIG. 228.—Congenital and acquired skeletal club hands. Operative results.

worn for a long time and the muscle power of the extensors must be carefully developed by manipulation, massage, and motion.

The treatment of the skeletal club hand deformity usually involves operative interference upon the forearm bones. A number of operative plans have been worked out and described. Bardenheuer in cases of radial club hand splits the ulna in a longitudinal direction inplanting the carpus between the two halves. McCurdy implants the proximal end of the ulna into the carpus after trimming the bone properly so as to bring ulna and center of carpus in proper position. Albee implants a tibial graft obliquely into the carpus and ulna attempting in this way to hold both bones together. The writer has used the implantation of the ulna into the carpus with very good cosmetic effect in one case of radial club hand in which the defect of the radius had followed osteomyelitis.

The Cleft Hand.—Synonym.—Lobster Claw Hand: Scheerenhand.

The deformity is very rare and only comparatively few cases have been described in the literature. A. H. Tubby reported a series of cases of cleft hand extending through five generations and associated with cleft feet. Cases reported by Bircher and others also showed a hereditary and familial tendency. The deformity is often combined with polydactyly, syndactyly, and other deformities. The cleft hand represents an extremity devided by a far-reaching cleft separating not only the metacarpals but penetrating into the carpus as well. It is usually associated with the loss of one or more fingers and their respective metacarpals. The condition is more often bilateral than unilateral and is frequently associated with cleft foot. One is often surprised to find the remaining fingers very limber and active so that the use of the hand does not necessarily suffer great restrictions. Two cases of this kind have come under the writer's personal observation. One was a child, nine days old when first seen, with bilateral cleft hand, the defect involving the middle and ring fingers of the hands. The feet showed similar clefts on both sides. The other case was eight months old when seen and had a lobster claw hand deformity of the right hand. Here the cleft seemed to be between the thumb and index finger. The latter, however, was abnormally large and the x-ray showed that this large index finger really represented a completely fused third and fourth finger, while the original index finger was totally missing. This case was also complicated by congenital absence of both tibiæ and congenital absence of both patellæ. Owing to the extremely good function shown by the cleft hands in both cases, no operative attempt was made to improve it.

Syndactylia.—Synonym.—Web Hand.

The web fingers may be divided in finger webs or cutaneous syndactyly; skin and fibrous connective tissue webs, and, lastly, bony fusions. Some cases show apparently completely fused fingers. On x-ray examination, however, one often finds that there is still a skeletal distinction of two fingers and the bony fusion is at least incomplete. It is not uncommon to see phalanges and metacarpals partially fused. Sometimes the fusion will involve only the proximal halves of the phalanges so that the distal part has the appearance of bifurcation. In other cases, again, fusion involves the distal parts of the fingers more than the proximal parts. This is the case in many instances of cutaneous fusion where the tips of the fingers are intimately grown together so that it becomes difficult to identify the individual fingers

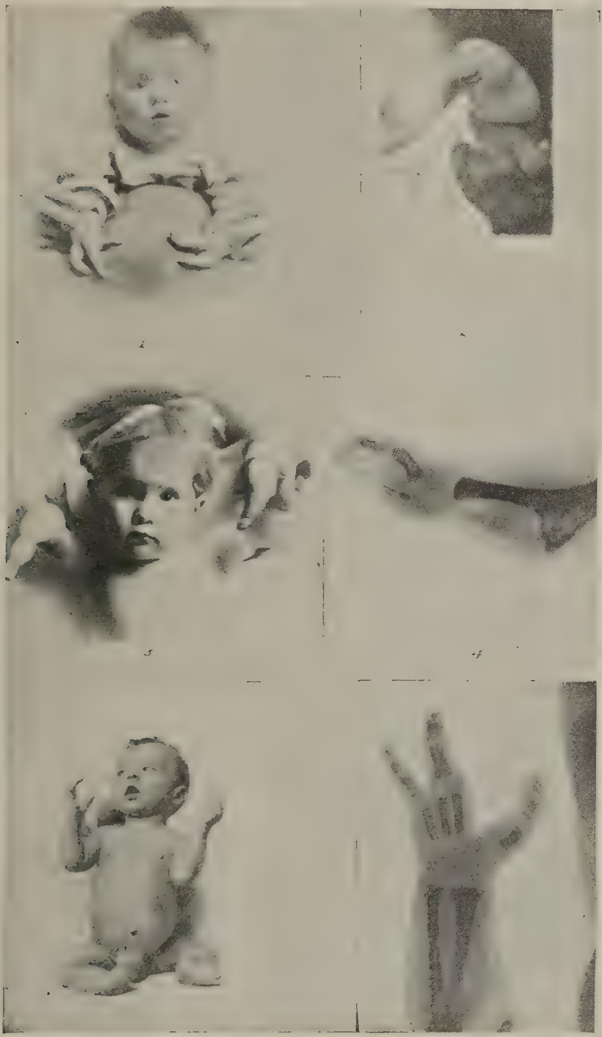

Fig. 229.—Cleft hand.

(acro-syndactyly). Combinations with other deformities are frequent. Syndactylia is often associated with ectrodactylia and ectromelia and is also often found in club hands and other congenital deformities of the fingers. It has also a strong hereditary tendency as proven by the very numerous reports of syndactylic families. One of the most striking instances is that mentioned by Luaces who reported a syndactylic family of 39 members among which were 14 cases of syndactylia.

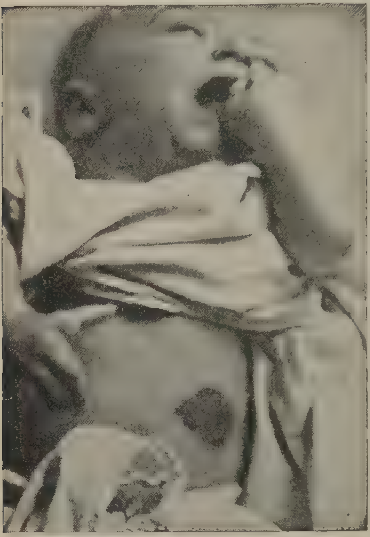

FIG. 230.—Cleft hand.

Treatment.—Several methods have been advised for the operative treatment of this deformity. The one most frequently used is that of Didot. This is a skin plasty carried out from dorsal and ventral incisions, one being on one and the other on the opposite side of the syndactylic web. From these incisions, flaps are raised which have their bases on opposite sides of the fingers dorsally and ventrally. The free ends of the flaps are then drawn around the adjacent sides of the finger so that a complete covering of the fingers results after separation. While this method is useful for a majority of cases, yet there are others in which the amount of skin obtainable is not sufficient for complete covering and which therefore call for other methods of skin plasty. In some instances of this kind one will find the Italian method of pedunculated flaps raised from the chest or abdominal wall extremely useful. There is no single method applicable for all cases and it is therefore necessary to individualize strictly. These plastic operations must be carried out with the greatest care and especial attention must be paid to painstaking adaptation, careful sewing, and, above all, to the question of vitality of the flap. The proper spread of the fingers must be secured and it is well to dress the cases several days after operation because of the wound secretion which is liable to cause laceration of the flap. Especially in cases operated by the pedunculated flap method a great deal of attention must be paid to the aftertreatment. The scars show a propensity to contract for a long time after operation. They should therefore if possible be laid on the dorsal surface of the finger and when a volar scar is unavoidable, the finger should be held in complete extension for a period of several months, and appropriately shaped finger splints should be used to secure this position. With the closure of the wound, the mechanical aftertreatment begins, and the first consideration must be the prevention of scar contractures by means already alluded to. Only by persistent aftertreatment which includes the passive and active extension movements of the finger as well as ab- and adduction, can the tendency to contracture be overcome. The web at the base of the fingers especially shows great tendency to contract and to invite recurrence of the webbing. When the pedunculated flap is used, the surgical principles governing the use of this flap in general

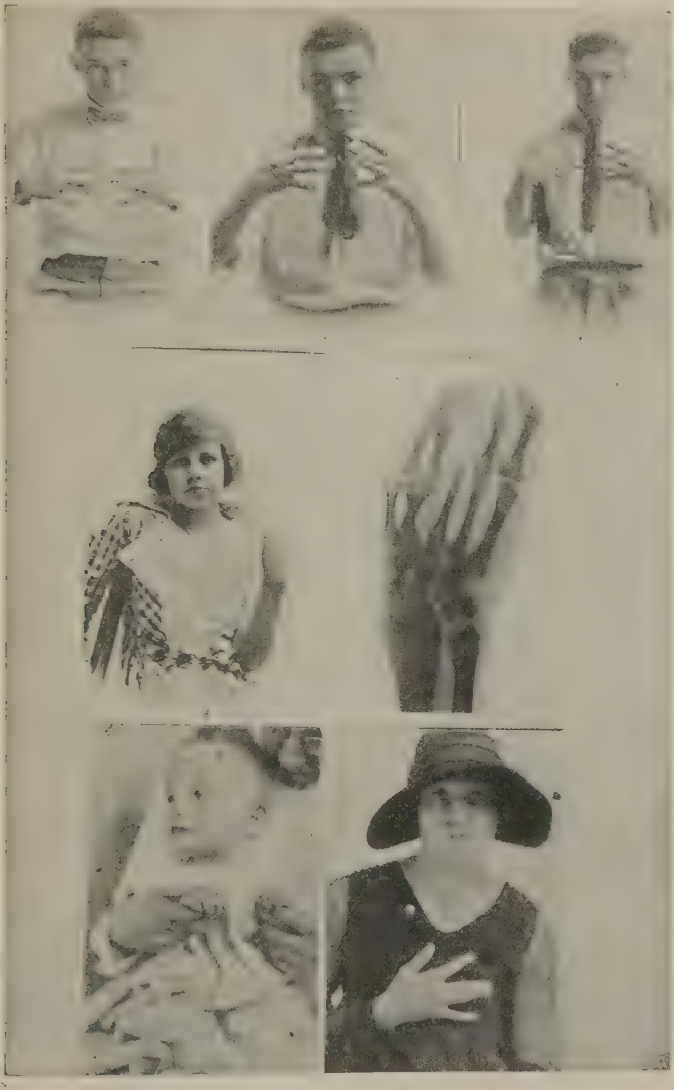

FIG. 231.—Syndactylia.

also apply in these instances. Foremost among these are the proper width of the pedicle, the proper anatomical position of the flap which will take in consideration the conditions of arterial blood supply to the skin, the avoidance of undue tension of the flap, and of twisting or tilting the pedicle.

Of eight cases operated by the writer, Didot's plasty was used in six, while the other flap methods were used in the remaining two cases. In cases where there was a volar web which drew the finger down toward the palm, the writer has repeatedly used a suggestion of Pieri who makes his incision in a zigzag fashion over the scar. This allows the raising of several flaps which may be drawn across over the crest of the contracting fold giving thereby not only ample skin for covering but avoiding also subsequent contracture.

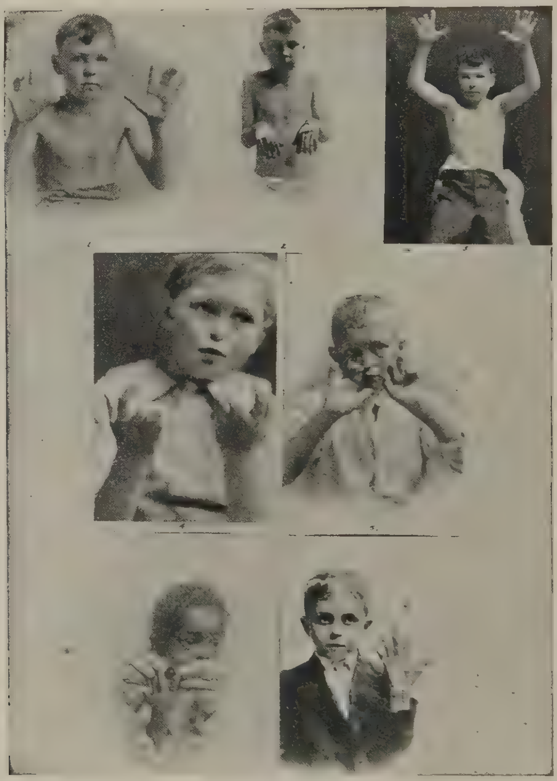

FIG. 232.—Syndactylia.

Symphalangism.—The deformity is very rare. It is characterized by the fusion either partial or complete of the interphalangeal joints. This fusion is not always a true bony fusion but often represents merely extra-articular contractures of the ligaments or tendons.

The *x*-ray picture readily reveals cases in which fusion is only apparent by showing a distinct separation of the phalanges and a clear joint space. True fusion is very uncommon, and only a few cases of this kind have been reported. In these cases the *x*-ray shows the phalanges actually fused and there are no traces of a joint space. The writer has observed two cases, both occurring in members of the same family. The first case was a boy three years old who had bilateral, cutaneous syndactilies between the second and third fingers. The *x*-ray showed true bony fusion between the basal and middle phalanges of the fourth and fifth fingers, that of the fifth finger being more complete than that of the fourth. This deformity also was symmetrical on both hands. The sister of this patient, six months old, showed absolutely identical deformity except that the fusion of the fourth fingers on both hands was more complete than in the case of the brother. In this second case there was also a fusion of the radius with the humerus on the right side.

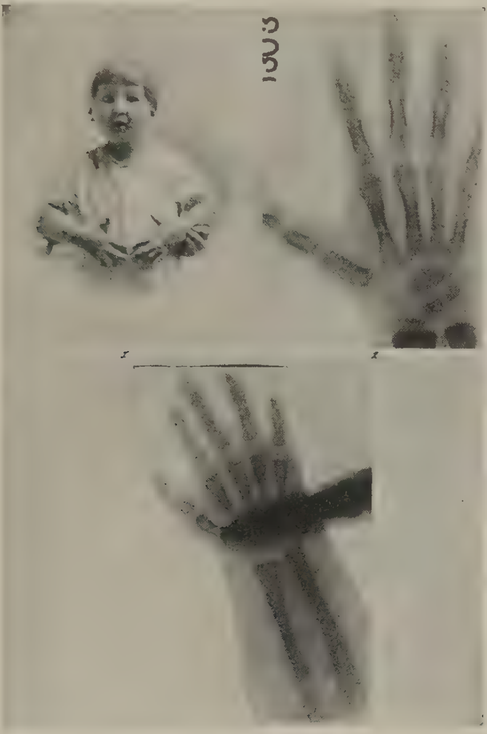

FIG. 233.—Symphalangism.

Ectrodactylia.—Ectrodactyly is a congenital absence of one or more of the entire fingers with their metacarpals. The cleft hand which has been described in a previous paragraph is a type of ectrodactyly. One may distinguish a marginal and a central form. In the marginal form the fingers of the radial or ulnar border are suppressed while the middle fingers remain. In the central form, there is a defect of the middle fingers and their metatarsals, and these are the cases which result in cleft formation of the hand.

A total ectrodactyly is a condition in which all fingers are missing except for small stumps. These stumps often carry osseous nodules and may articulate with the remaining metacarpals or in cases of metacarpo-phalangeal ectrodactyly the metacarpals also may be suppressed. A case of ectrodactyly has been observed by the writer. The patient, a boy three years old, showed the deformity on both hands. On the left hand there was present a thumb and one finger; on the right hand, the two central fingers were present while both the thumb and little fingers were missing. There was also a total absence of the ulna and complete fusion between radius and humerus on the left side, and complete absence of the radius with fusion of the ulna and the humerus on the right side.

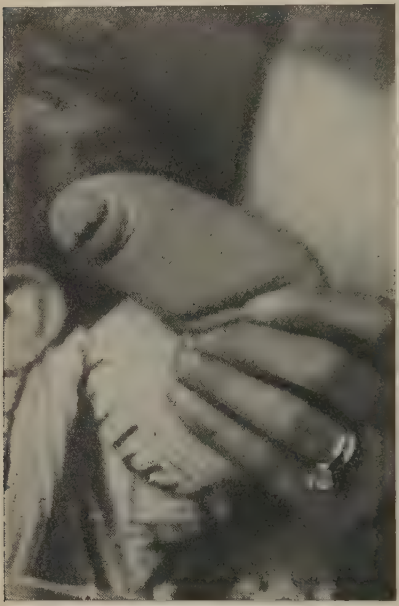

Fig. 234.—Ectrodactylia.

Polydactylia.—In polydactylia also a central and a marginal type may be distinguished. Polydactylia may be produced by bifurcation of fingers over the metacarpo-phalangeal joints, in which case there is a total duplication of fingers; or from a bifurcation of the metacarpal bones or of the phalangeal bones themselves. In the latter instances, there is an approachment to the syndactylic forms with complete fusion. The marginal type, that is, the duplication of thumb and little finger, is by far the most frequent form. Heredity is a distinct feature of this congenital deformity. Numerous reports of polydactylic families are on hand. Bernard's case covers five generations. Brandeis reports on a whole family with hexadactyly. The largest collection of familial deformities of this type is that of Moore who in a family of seven generations found among 237 members 20 cases of polydactyly.

The supernumerary fingers are at times well developed while in other instances they may be extremely rudimentary. But even these small rudimentary appendices may contain their own little tendon and may even present all the elements of a normal finger in a rudimentary and abortive way.

Polydactylia is often combined with other deformities. The writer has observed a case of polydactyly in a boy two years old in which the thumb was duplicated on the right hand with all distinct phalanges of the supernumerary thumb. In another case the patient showed a simple rudimentary little finger attached to the ulnar border of the hypo-thenar eminence. It was so small that it could be taken for a cutaneous wart, but it had to be recognized as a supernumerary finger because it carried a tiny little nail and upon removal it was found that it also had a perfectly separated minute flexor tendon. There are numerous variations and degrees between these two extreme cases mentioned.

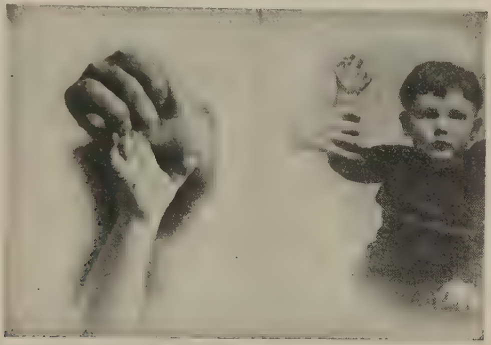

Fig. 235.—Polydactylia.

Congenital Contractures of Hand and Fingers.—Amniotic adhesions and amniotic furrows play a very conspicuous part in congenital contractures of hand and fingers. Furrows are very often seen encircling the fingers, thumb, hand, and wrist, and the constriction always leaves a more or less deep groove. Not infrequently the constriction goes on to complete amputation. In a case of this kind observed by the writer, it was the index, third, and fourth fingers of the left hand which were furrowed. Motion was in no way restricted as the groove did not cut down deep into the structures. The amniotic nature of this deformity was further evidenced by the fact that the child had a spontaneous amputation of the right leg at the lower third of the tibia, and there was absence of the end phalanx of the second toe of the left foot. In another case of this type, several furrows were seen on some fingers together with complete amputation in the basal phalanges of others and deep amniotic furrowing of the tibia.

Little can be done as a rule to mitigate this deformity. It is possible however to excise the furrows if they are not too deep and to bridge the defect by skin plasty. In one case in which this was done by the writer, a considerable cosmetic effect was obtained.

Thumb and fingers are often involved in a flexion contracture produced by volar webs. These webs are cutaneous and not unlike those seen after burns of the palm of the hand except that they are congenital and they represent intact and normal skin. They are not identical with the contractures seen in contracted club hands because of the prominence of the webs and of the intactness of the deeper structures. It is quite probable that here too amniotic adhesions are at work. In one case in which the fracture involved the little finger of the right hand the picture resembled somewhat that of Dupuytren's contracture except that the deformity was congenital and not progressive. In another case, a boy 12 years old, the fifth fingers of both hands also were held in flexion position by a web extending from the middle phalanx to the base of the hand. This case was operated on by skin plasty and the deformity was corrected. In a third case, a girl 1½ years old, the congenital contracture involved the second, third, and fourth fingers of the left hand, but in this case also the deformity was

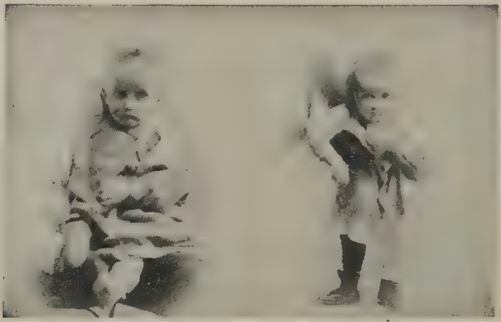

FIG. 236.—Amniotic furrowing of fingers and amniotic amputation of leg.

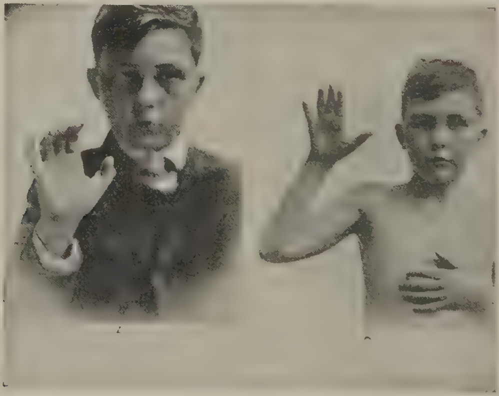

FIG. 237.—Congenital contracture of thumb.

corrected by skin plasty. In this latter case, the fact of importance was that there were notable deformities of the spine and trunk. There was a wedge formation of the fifth dorsal vertebræ, there were numerous fusions and anomalies of the ribs on both sides and there was also a congenital elevation of the scapula. This case shows that the deformity of the hand must have had a cause lying deeper than the external agencies of amniotic adhesions, and the development of the deformity must have reached much farther back into a very early period of embryonal life.

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ACQUIRED DEFORMITIES OF THE HAND AND WRIST

Madelung's Deformity of the Wrist.—Credit for the earliest observation of this unusual deformity is due to Dupuytren, who in 1839 described this condition in workers and especially in those whose work required straining of the wrist such as pressers, clothesworkers, teamsters, washerwomen, etc. The classical description of this disease by Madelung dates back to 1878. The characteristic symptom of this deformity is a bend in the lower end of the radius which deflects the hand toward the volar side and at the same time causes the ulna to become more prominent upon the dorsal side of the wrist. The anteroposterior diameter of the hand is increased and there is sometimes a slight lateral deviation of the carpus in addition to the volar deflection.

The flexor tendons of the wrist, especially the flexor carpi radialis and the flexor pollicis longus, come distinctly into relief. The relation of the radius to the carpus remains normal and there is no abnormal mobility in this joint. The dorsiflexion of the hand is impaired while the volar flexion is normal or even increased. The change of relation between ulna and the carpal bones is due to the fact that the ulna, not following the forward incurvation of the radius, becomes dorsally displaced so that a secondary dislocation in the ulno-cuneiform articulation is produced.

The changes in the configuration of the lower end of the radius are accompanied by pain which is especially noticeable in the formative stage of the deformity. In fact, it is usually the pain which calls the attention of the patient to the deformity. This pain comes on gradually and insidiously with the deformity and it becomes more noted upon exertion. As the deformity becomes more and more established, however, the pain gradually subsides.

Heredity seems to play a certain part in the deformity since among 85 cases mentioned by Estor, it was observed in 11 cases. Fere reports a case of a mother and two daughters afflicted with this deformity, and Jagot found it in three generations. Sometimes occupational strain seems to have an influence upon the formation of this deformity while in other cases rickets appear to have a certain etiological importance.

In a very complete review of the literature, Stetten in 1909 reviews 64 cases, 62 of which showed the typical deformity with the bend of the lower end of the radius toward the volar side. In two cases, however, the bend of the radius was backward causing a silver-forked deformity similar to that seen in Colles' fracture. Madelung's deformity is essentially an affliction of the growing age. The youngest case reported was eight and the oldest 25 years of age. The deformity has never been found to be congenital. It is now generally assumed that the important factor producing the deformity is a disturbance of growth in the lower end of the radius, and this disturbance aided by the pull of the flexor muscles develops the characteristic bend. The average time for the full development of the deformity is three years. The early or formative stage is associated with pain and might be considered the acute stage of the disease. Following this, the pain subsides, to disappear entirely with the full development of the deformity. In most of the cases, there is no great impairment of the function of the hand. Generally, however, the patient complains of a certain weakness, tires easily, and often has attacks of pain upon exertion.

From the viewpoint of differential diagnosis, there should be considered (1) Colles' fractures with forward displacement of the hand. This condition however can easily be established by the history and by the x-ray findings. (2) In some cases, arthritic conditions of the hand are apt to produce a backward dislocation of the ulna together with the volar contraction of the wrist. This condition is also easily differentiated because of the multiplicity of joint involvement, the history, and the distinct radiographic changes of arthritic nature.

Treatment.—Attempts to correct the deformity by braces or splints are of little value except that the pain may be alleviated by such contrivances during the early stages.

The only method which has proven successful in correction of the deformity is the osteotomy. A wedge osteotomy was used by Redard. Duplay and Kirmisson used a simple osteotomy, and Poulson the oblique osteotomy for correction of the deformity. Occasionally the bend in the radius is not confined to the lower end but extends through the entire length of the bone, a condition which has been especially described as radius curvus. In cases of this kind osteotomy gives less satisfactory results, but in an ordinary type of sharp forward kinking of the radius, the operative results reported have been very satisfactory.

Following the operation, the hand is immobilized until solid union has occurred. After consolidation of the fragments, some medicomechanical measures of massage and exercises are instituted as are indicated in cases of fractures.

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Dupuytren's Contracture.—A description of this deformity has been given by Astley Cooper but it was Dupuytren in 1831 who first described a case of this type of contracture, and following his description the deformity has since assumed his name. The condition is essentially a thickening and contracture of the digital processes of the palmar fascia. Following the involvement of the digital processes, the main body of the palmar fascia also becomes involved. The result of this thickening and contracture of the fascia is that the fingers become gradually drawn into the palm. The flexor tendons themselves are not involved and take no part in the contracture. The deformity begins slowly and insidiously. There are no signs of inflammation, there is no tenderness. It is usually the fourth and fifth fingers which are involved. They appear to be drawn toward the palm of the hand by a strong firm cutaneous fold in which the tough fibrous tissue of the thickened fascia can be felt. That the flexor tendons of the fingers take no part in the contractures can be shown by the fact that in extreme flexion of the wrist there is no release of the contracture, but it remains the same whether the wrist is flexed or extended. Microscopically, the thickened palmar fascia consists of fibrous strands intermingled with cellular infiltration. The skin covering the thickened portions is at first normal, but later it becomes puckered and thickened as the fibers of the palmar fascia which adhere to the skin begin to contract. Pathologically, the condition can be classified as a localized fibrositis with increased formation of connective tissue fibers. This proliferation is most marked around the arterial sheaths and the subendothelial layers of the capillaries.

Block who collected 240 cases in the literature found 104 bilateral and 130 unilateral cases. Of the unilateral cases, 89 involved the right and 47 the left hand. The statistics of Hume show a similar preponderance of the unilateral cases over the bilateral and of the right hand over the left hand. The statistics of Keene also show similar relations.

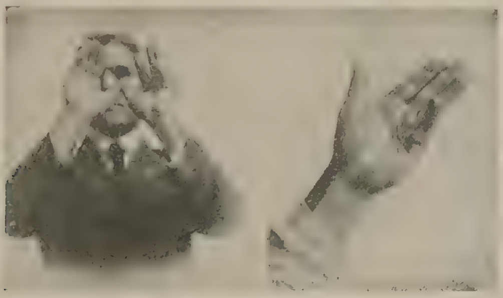

FIG. 238.—Dupuytren's contracture.

Treatment.—Conservative treatment may be tried in earlier stages. It consists in extension, manipulation, massage, application of heat, Brier's hyperemia, and injections of fibrolysin.

Advanced cases, however, and especially those showing contracture in full development, must be treated surgically. Different methods have been applied. Adams performs multiple tenotomies first near the carpus, then at the metacarpo-phalangeal joints gradually severing all strands and extensions of the contracted fascia. He begins his mechanical aftertreatment four to five days following the operation.

Tubby makes a longitudinal incision over the most prominent portion of the palm to which a number of cross-incisions are added. Flaps are dissected and turned back. In so doing one must avoid the buttonholing of the skin. Each portion of the fascia is dissected and hemorrhage is arrested by pressure and hot water. Fibrolysin is then poured into the open wound and rubbed in thoroughly for two minutes. The hand is put up in a malleable splint for two weeks. Hutchinson advises the following procedure: (1) The strands of the contracted and thickened fascia are dissected from a palmar incision, then a resection of the head of the basal phalanx is done from a semilunar incision laid dorsally over the first interphalangeal joint. This author strongly opposes the use of splints because of its stiffening effect upon the joints of the affected fingers.

One of the most thorough methods is that of Bruce Gill. This author makes a transverse incision over the distal palmar crease. Through this incision a careful dissection is made of the entire palmar fascia to or beyond the crease at the base of the thenar eminence. Distally, he carries the dissection to the web of the fingers, carefully avoiding buttonholing of the skin. He then excises the contracted fascia being duly careful not to injure the underlying nerves and vessels. If the first interphalangeal joint cannot be extended, he then excises the head of the basal phalanx following Hutchinson's procedure. Then a small free transplant of the thigh is inserted smoothly between the palmar skin to prevent adhesions of the tendon.

All operative treatment must be followed carefully by extensive aftertreatment consisting in careful and continuous splinting, massage, passive, and active motion, and application of heat.

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Trigger Finger.—Synonyms.—Snapping Finger; Doigt a Ressort. The snapping or trigger finger is occasionally seen in children. An extensive report of this deformity is that of Hiller (1908) who collected 161 cases in the literature. Of these cases, 41 involved the thumb, 47 the middle finger, 12 the index finger, and 44 the ring finger.

The pathological conditions underlying this deformity might be divided into three groups: (1) Changes in the tendon; (2) changes in the tendon sheath, and (3) changes in the capsule and the capsular ligaments. Changes in the tendons consist in tendon duplications, tendinous bulbs, fibromas of the tendon, or hyperplasia of the tendon tissue. Changes in the tendon sheath may consist in hypertrophic villi of the sheath, or fibromas of the sheath or of foldings and duplications of the tendon sheath. Capsular and ligamentous changes are either cartilaginous ridges within the joint or changes in the lateral ligaments of the interphalangeal joints.

The principal symptom of the snapping finger is a snapping sign which occurs when the finger is flexed at a certain angle. From this point on further flexion of the finger is carried out with difficulty only and with a distinct effort which is accompanied by an appreciable jar or snap. At the moment of snapping, pain is felt in the palm of the hand. Often the other hand is necessary to accomplish flexion beyond the critical point. In extending the finger from full flexion a similar difficulty arises again at the same angle. Here also a distinct effort is necessary to overcome the obstacle and there again appears a characteristic jerk and often an audible click.

If the obstacle is not great, the motion remains painless. Often, however, and especially in the initial stages, there exists considerable pain. On the volar surface of the metacarpo-phalangeal joint of the finger involved, there is often noticed a sensitive tumor of the size of a millet or pea. In one case observed by the writer, the snapping finger was seen as a congenital deformity in a young infant a few months old. There was a slight thickness of the tendon of the index finger on the volar side near the metacarpo-phalangeal joint. Motion was painless and the obstacle gradually disappeared within a few months when the motion of the finger returned to normal.

Treatment.—In milder cases the treatment consists in manipulation and splinting and the application of massage. Cases which do not yield to these measures require operative interference. The operation consists in an incision over the thickened tendon, in the opening of the tendon sheath, and in the removal of the cause of constriction. Hildebrandt reported on operative procedures of this deformity in which spindle-shaped thickenings of the tendons were removed with the resulting cure. Poulson who compiled statistics covering operative or necropsy findings of this deformity found among 64 cases examined changes of the tendon itself in 26 cases, changes in the tendon sheath alone in 13 cases, changes in both tendon sheath and tendon in 18 cases, and changes in the joint itself in six cases.

Tendon Ruptures.—The subcutaneous ruptures of tendons are rare. The tear usually occurs at or near the insertion of the tendon or at the junction between tendon and muscle. The so-called spontaneous ruptures of the tendons are hardly ever purely traumatic. Investigations have shown that the tendons ruptured are almost always diseased. The tendons consist mainly in hyaline degeneration of the tendons, subsequent to chronic changes in the joint, especially arthritis deformans.

The ruptures of the finger tendons involve mostly the extensors and especially the extensors of the thumb. According to Schlatter's observation a comparatively slight trauma often suffices to cause rupture. Three cases of rupture of the long extensor tendon of the thumb are reported by Van Verth, two occurring in waiters and one in a wood carver. To this group of tendon ruptures, there belongs also the so-called drummer's palsy which is in effect a rupture of the long extensor of the thumb induced by continuous strain.

In ruptures of the tendon, the ends appear frayed and the microscope shows round-cell and spindle-cell infiltration in the tendon with hyaline necrosis in the center. Numerous hemorrhages are also seen in the tendon tissue.

The injuries leading to tendon ruptures are often occupational in character, for instance, buttoning of tight buttons, pulling of fingers wrestling, basket or baseball playing; bowling, etc., may cause this injury.

The typical symptom is inability of active extension of the phalanx although passive extension is entirely free. The end phalanx drops into a position of flexion (drop phalangette). In the majority of cases immobilization restores the ability of active extension although the prognosis of the conservative treatment is not always good and operation is sometimes required. The operative procedure is simple. The proximal end of the tendon can be picked out easily and fastened to the distal phalanx of the finger. Only in older cases the place of rupture will not be so easily distinguishable and one may have to resort to the expedient of fastening the proximal end to the capsule of the interphalangeal joint when the distal end cannot be isolated.

Dislocation of the Extensor Tendons of the Fingers.—Dislocation of the extensor tendons is extremely rare. Schlatter's statistics show that only 10 cases were reported in the literature up to 1909. In dislocation of the tendon, the two halves are displaced laterally. Consequently, active extension is impossible at the start; however, if the finger is helped at the beginning of extension, the remainder of the motion can be carried out actively. The treatment of these cases consists in the suturing of the two laterally displaced halves of the tendon.

Fractures and Dislocations to Fingers.—The typical dislocation of the phalanges is that of dorsal dislocation of the end phalanx. Dislocation in the interphalangeal joints is much less frequent. Reports of Guibe on metacarpo-phalangeal and interphalangeal dislocations contain 84 dislocations of the metacarpo-phalangeal joints of the thumb, 27 of the metacarpo-phalangeal joints of the fingers, 26 of the proximal interphalangeal joints of the fingers, and 69 of the distal interphalangeal joints of the fingers.

Of great interest are dislocations of the thumb. The classification of Faraboef distinguishes between complete and incomplete dislocations on one side and the so-called complex dislocations on the other. In the incomplete dislocations, the sesamoid bone still adheres to the joint surface of the metacarpal. In the complete dislocations, the sesamoids are displaced upward together with the distal phalanx. In the complex dislocations, they are wedged in between metacarpal and basal phalanx.

The most frequent form is the posterior dislocation caused by fall upon the extended thumb or by blow. This dislocation is accompanied by a tear in the capsular ligament as well as in the lateral ligaments. According to Guibe, the displacement itself may be complete or incomplete. In the incomplete displacement, the distal phalanx rests with its articular surface upon the head of the basal phalanx at a more or less right angle. In the complete displacement, the distal phalanx is pushed entirely over the head of the basal phalanx so that the axes of these two bones are parallel.

The dislocations become irreducible within a very short time, in fact, within a very few days after their occurrence. This is due partly to the displacement of the extensor tendon and partly to the interposing of fibrous and tendinous tissue between the articular ends. In reducing the dislocation, simple flexion suffices for the incomplete type. In complete dislocation, however, traction and pull forward is necessary together with hyper-extension. When reduction becomes impossible, open operation is indicated. A flap incision is made over the dorsal side of the articulation, the convexity of the flap pointing distally. The extensor tendon is carefully dissected and drawn aside. The resection of the interposed capsular tissue is now possible. It is also often necessary to resort to resection of the head of the basal phalanx in order to accomplish the reduction.

Forward dislocation of the thumb is much less frequent than posterior dislocation. Here too complete and incomplete dislocations may be distinguished. In the complete dislocation, the distal phalanx is dislocated upward as well as forward and the axes of the bones are parallel. In the incomplete dislocation, the distal phalanx rests with its posterior border at more or less right angle upon the head of the basal phalanx. This dislocation also becomes irreducible within a few days and then operative interference is necessary.

Dislocation of the fingers usually is caused by fall or blow upon the finger which forces the joint into hyper-extension and results in posterior displacement of the distal portion. Lateral displacement of the fingers by direct violence is not infrequent and is usually accompanied by fracture sprains of the bases of the distal phalanx. The reduction of all dislocations of this type likewise becomes impossible after a short period and operative interference is then required.

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CHAPTER CXVIII

DISEASES AND DISORDERS OF THE KNEE

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The affections of the knee joint encountered in childhood are many, and their exact diagnosis is not always easy notwithstanding the fact that the knee joint is a structure of easy approach and broad exposure. The large variety of afflictions makes it necessary to be accurate and explicit in the definition of the trouble and a good knowledge of the regional anatomy is one of the prerequisites of accurate diagnosis.

ANATOMY

The apparently complex structure of the knee joint is best understood if one starts from the conditions as they prevail in some of the lower animals where this joint is actually a twin joint, each half embracing one condyle of the femur and the corresponding one of the tibia. In the human knee the same conception may be entertained only with the difference that there exists a wide communication in the medium septum of the knee by means of a large central opening (J. H. Prentiss). This central partition is formed by two projections of the synovial membrane. The anterior projection running backward is formed by the folding of the synovial membrane over the fat-body which lies behind the patellar tendon. In so folding, the synovia raises a median ridge which is known as the ligamentum mucosum, and also two lateral ridges which diverge into the joint and which are known as ligamenta alaria.

The posterior part of the median curtain is caused by a folding of the posterior portion of the synovial membrane over the crucial ligaments which really represent reinforcements of the posterior capsule.

The space between these posterior and anterior curtains represents the communication of the two halves of the knee joint. Anteriorly, the central portion of the joint is formed partially by the articulating surface of the patella. Upward and underneath the quadriceps tendon, the knee-joint cavity is continued by a bursal sac known as the subfemoral or suprapatellar pouch. The capsular insertion follows largely the outlines of the cartilage covering of femur and tibia except in the midline where the median folds just described are projecting between the condyles of the femur and that of the tibia.

The bursæ surrounding the knee joint are numerous, the most important being the *suprapatellar* pouch which in the vast majority of cases is really a continuation of the knee-joint cavity. The bursa lying between the patella and the skin is known as the *prepatellar* pouch, and the third bursa, lying between the ligamentum patellæ and the tibia, as the *infrapatellar* pouch. The two latter bursæ do not communicate with the joint. Posteriorly, there are bursæ connected with the tendons of the biceps, the popliteus, the inner head of the gastrocnemius, the sartorius, the gracilis, the semi-membranosus, and the semi-tendinosus muscles.

The *motion* of the knee joint is principally that of flexion and extension in the sagittal plane. In the normal knee, a slight rotation inward occurs at the beginning of the flexion due to the action of the inner hamstring muscles which insert forward into the upper portion of the tibia. There is also a corresponding slight outward rotation of the leg at the close of extension. The flexion motion in the knee joint itself is accomplished between the gliding surfaces of the tibia and femur with the aid of the two semi-lunar cartilages which even up the incongruences between the articular surfaces of tibia and femur, and which, therefore, have a certain limited amount of motion themselves. The radius of the rotation about the condyles of the femur is not constant, being larger in the anterior and smaller in the posterior portion of the curve of the condyle, the ratio of the anterior to the posterior radius being as 9:5. This arrangement is justified by dynamic reasons. It also presupposes a considerable amount of give on the part of the anterior portion of the joint, and this again must be safeguarded by a rather complicated apparatus of reinforcements in order to keep motion within physiological limits and to protect the joint against strain and stresses.

The checking of the motion of the knee joint is carried out largely by the ligamentous reinforcement of the capsular apparatus. Anteriorly, the tendon of the quadriceps and the ligamentum patellæ check the joint against hyperflexion. Lateral motion is checked by the internal and external lateral ligaments. Inasmuch as these ligaments run also somewhat posteriorly to the axis of the knee joint, they are also checks against hyper-extension. This can be seen from the fact that the internal lateral ligament is inserted into the posterior portion of the inner condyle of the tibia while the external, divided into a short and long portion, fastens to the head of fibula. Anterior and posterior dislocation of the tibia are checked by the crucial ligaments which are also checkers of extreme flexion and extension, the anterior becoming taut in extension and the posterior in flexion of the knee. The iliotibial band is also of importance for the stability of the knee as it checks against lateral motion and protects the knee joint against outward thrusts.

CONGENITAL DISLOCATION OF THE KNEE

Congenital dislocation of the knee joint is a very rare occurrence. According to Kroenlein, there are seen 90 cases of congenital dislocation of the hip to one case of congenital dislocation of the knee. One of the most complete articles on this subject is that of Drehmann in 1900, who collected 127 cases in the literature.

Much discussion has arisen regarding the identity of the condition. Phocas and Potel considered many cases reported as congenital dislocation to be simply cases of congenital genu recurvatum, finding true congenital dislocation only in a small minority of the cases reported. On these grounds, Mayer opposes the term genu recurvatum as well as that of luxation of the knee, calling the condition a partial dislocation or subluxation of the tibia upon the femur. In Drehmann's statistics, there were 54 unilateral cases of anterior subluxation and 48 bilateral cases.

All cases of anterior dislocation show as their cardinal symptom a considerable degree of hyper-extension of the knee sometimes to the point of actual contact between the anterior surface of the leg and that of the thigh. The leg is usually slightly flexed in the hip joint and the toes of the afflicted limb touch the abdominal or inguinal region. Motion of the knee joint is only possible in direction of further hyper-extension.

On the anterior surface of the knee joint the skin is redundant, showing several transverse folds. Behind, in the popliteal region, there is a prominence over which the skin is tight. This prominence corresponds to the condyles of the femur which are entirely palpable in their posterior aspects including the intercondyloid fossa. The joint line of the tibial head cannot be made out by palpation. Underneath the anterior folds of the skin, the movable patella may be found. Further hyper-extension may be carried out without resistance. But actual flexion or sometimes even a decrease of the hyper-extension position is inhibited, the efforts meeting with a peculiar spring-like resistance. This resistance is often furnished by the contracted quadriceps muscle although there are a number of cases in which the absence of such a contracture has been expressedly noted. Lateral motion of the knee is not possible. In very young patients it is often possible by traction to diminish the hyper-extension and even produce flexion of the knee, but in most of these cases one will note that as soon as traction ceases the limb will immediately resume its former position of hyper-extension. When the child is several months old, it is commonly observed that the original hyper-extension diminishes to a degree which is probably due to the weight of the leg itself tending to overcome the hyper-extension. Gradually there develops a condition which parallels a traumatic dislocation. The displacement of the leg is forward and when attempts at standing have been made, there is then added an abnormal lateral mobility of the joint, and often even a flail joint becomes established. The patella is usually displaced upward and often it is very rudimentary in its development. However, statements of total absence of the patella should be taken with caution because the bone may be very small and may escape detection by palpation.

Mayer in 1912 reported 21 observations made on specimens from the Munich Pathological Institute. He finds that the lesion consists in the subluxation of the tibia forward and upward upon the femur and in a corresponding shortening of the extensors of the knee as well as of the anterior capsule. The flexors of the knee in these cases are dislocated forward in front of the axis of the knee joint so that these muscles really appear to be converted into extensors of the knee. The patella is usually displaced upward. It is interesting to find an articular surface placed at the anterior aspect of the femur running from below upward and forward, and upon this surface the posterior surface of the tibial head articulates.

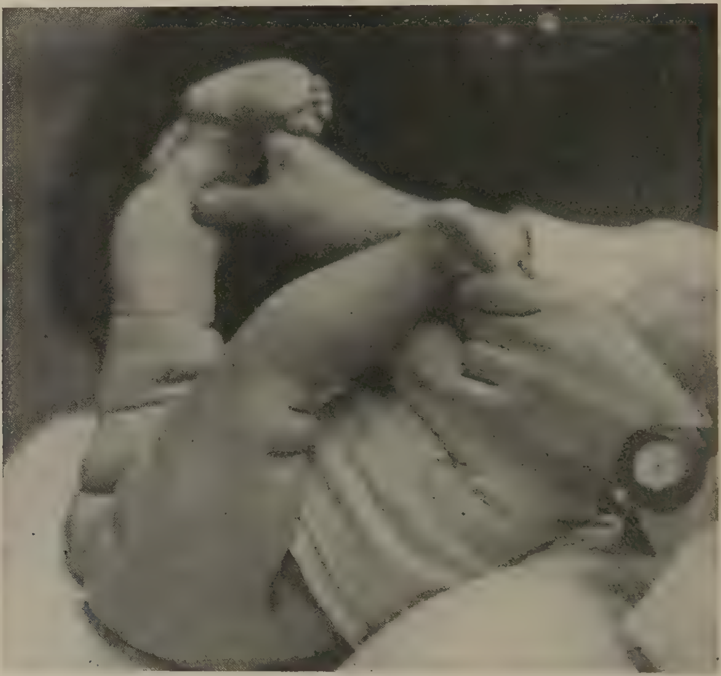

FIG. 239.—Congenital dislocation of knee.

Nothing is definitely known as to the etiology of the condition. The consensus of opinion seems to be that the disorder is mechanical in nature. Miller believes that the abnormal extension position is caused by the pressure of the uterine wall when the anmios fluid is scarce. This opinion is strongly criticized by Patel who in turn believes that primary contracture of the extension musculature is the principal agent. However, it seems that the most plausible explanation is that of a forced position in utero which produces at first simple over-extension of the knee with a genu recurvatum and which later causes the gradual displacement of the joint surfaces. Many children born with congenital subluxation of the knee also show other deformities such as congenital dislocation of the hips and, in some cases, there is observed a general relaxation of almost all joints such as in a case cited by Drehmann.

Treatment.—The principal obstacle to reposition in young children is not so much the shrinkage or contraction of the structures of the anterior surface of the knee and thigh as is the fixation of the tibia upon the anterior surface of the condyles of the femur. Attempts at straightening cause the tibia to be lifted off from its bed at the anterior condylar surface so that this maneuver does not reduce the dislocation though it may cause an apparent correction of the deformity.

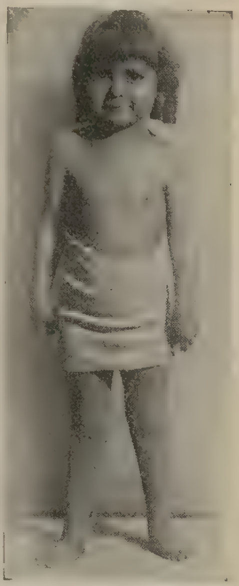

Fig. 240.—Congenital dislocation of knee. Case of Fig. 239 after operative correction.

The bests results are obtained in cases which are treated at the earliest possible moment. The reposition maneuver should be traction, then increase of the hyper-extension, and then gradual flexion. In newborn children, or in children a few weeks old, such reposition may be accomplished without anesthetic, and the gradual correction may be obtained by application of a series of casts or of splints. In older children (several months old or older), the tone of the extensor muscle is such that even after reduction a forward dislocation of the tibia is very likely to recur. It is necessary in securing the positon obtained after the reposition maneuver to exert proper forward pressure upon the condyles of the femur as well as pressure backwards at the tibial head. A circular plaster cast holding the knee in the best possible position of flexion will be the surest way of immobilization. Children who already walk should remain in the cast with the knee in moderate flexion, and should continue to walk in order to encourage the functional reformation of the joint bodies.

Cases not amenable to bloodless reduction, and these are all older cases or those in which the obstacles in front of the knee joint are unusually severe, must be treated operatively. Mayer makes a longitudinal incision into the patellar and quadriceps tendons, dividing the tendons above and below on opposite sides similar to what is done in lengthening the tendon of Achilles. He found in his case that it was possible by forward pressure upon the femur to overcome the hyperextension. Reduction of the subluxation was accomplished with a distinct click as the tibia slipped over the prominent lower border of the femur. Where the anterior capsule is sufficiently elastic to accommodate itself to the new position, nothing further need be done. But where this is not the case, the forced flexion of the knee joint will leave the gap in the anterior capsules which may have to be covered by a fascial plasty.

The author has observed a case of bilateral congenital subluxation of the tibia in which operative interference appeared necessary after repeated attempts at bloodless reduction had failed. The case was that of a female child six months old which had been born with an ulnar club hand, congenitally flat feet and double congenital subluxation of the knees. Operation was performed on both knees. On the right knee the quadriceps tendon was divided from a curved flap incision, the knee joint opened, and reduction was obtained. Only after dividing the lateral portions of the capsule was it possible to flex the knee to right angle. There was a large gap in the anterior aspect of the knee joint which necessitated a fascia plasty, the fascia being taken from the thigh and being implanted into the edges of the wound in the anterior capsule. On the other knee a similar operation was performed and fat was used to cover the defect in the anterior capsule. On both sides, the operation resulted in the formation of a serviceable knee, the coexisting genu valgum deformity being later corrected by osteotomy. At present, the child is able to walk without difficulty, but the knees are still being secured by braces.

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CONGENITAL DISLOCATION OF THE PATELLA

The congenital dislocation of the patella may be either partial or complete. The usual form of dislocation is outward. Upward displacement is rare. A thorough collection of this deformity was made by Steindler in 1898 who reported 67 cases in the literature, of which 64 cases were outward dislocations and three inward dislocations of the patella. When the knee is extended, the patella is usually in place becoming dislodged upon flexion. The displacement is sometimes such that the lower end of the femur is uncovered and becomes palpable underneath the skin. Occasionally a genu valgum deformity is seen in cases of congenital dislocation of the patella. In a case reported by Spitzy, the patient was a girl 10 years old who showed a marked degree of genu valgum deformity. The patella was situated on the outside of the outer condyle and was so far dislodged that the ligamentum patella pointed downward and inward. Of the portions of the quadriceps muscle, the vastus externus and rectus cruris were well developed, but the vastus internus was atrophic and was represented only by a thin muscular strand running toward the inner side of the tuberosity of the tibia. The knee was held in flexion. Upon attempts to extend the knee, the already existing outward rotation of the leg became decidedly increased, and instead of extension, flexion of the knee to about 30° resulted from the contracture of the muscle. This is explained by Spitzy very thoroughly on the grounds of the changed dynamic conditions under which the quadriceps muscle acted in this case.

In regard to the function of the knee joint, in cases of congenital dislocation of the patella, the reports vary. Some cases are reported as showing very grave functional impairment. On the other hand, in the case of Uhde, the patient had good use of his limb, was very fond of dancing, and experienced no difficulties in doing so. It seems that the functional disturbances depend largely upon the condition of the ligamentous apparatus of the knee and not only upon the actual change of anatomical alignment.

Treatment.—The secondary changes in the knee joint such as flexion contractures and valgity may be overcome by tenotomies and forcible extension under anesthesia. The correction obtained is then to be retained by plaster-of-paris dressings which must be changed from time to time to add additional correction. La Roux suggests attaching of the ligamentum patella to the inner side of the tibia, while Heusner favors pleating of the capsule of the joint with the attaching of the semi-tendinosus tendon to the inner border of the patella similar to what is done in cases of habitual dislocation.

The upward displacement of the patella is usually seen in connection with congenital dislocation of the knee, and has already been described under this heading.

HABITUAL DISLOCATION OF THE PATELLA

Following traumatism, especially traumatic dislocations of the patella associated with injuries to the fibrous structures of the inner portion of the knee joint, there develops occasionally a habitual or recurrent dislocation of the patella. The displacement is almost always to the outer side. A short consideration of the normal anatomical conditions of the knee relative to the patella will be of advantage to the understanding of the mechanism of this disability.

Normally the slipping of the patella outward is prevented by the forward projection of the anterior surface of the external condyle of the femur. The lower attachment upon the femur of the vastus internus, as compared to that of the vastus externus, holds the balance against this bony check represented by the projection of the external condyle. If there is an outward displacement of the tibial tubercle, such as exists in the case of genu valgum, the tendency of the patella to outward dislocation is increased. If to this is added further a relaxation of the quadriceps muscle and a relaxation of the internal portion of the capsular ligament, it can be seen easily that in hyper-extensions of the knee, a dislocation may occur upon a forcible pull upon the patella outward not sufficiently counteracted by the natural safeguards against displacement. Consequently, we see this dislocation occur with preference in patients of valgity of the knee and especially in young girls. At the moment of dislocation there is a distinct and violent pain which causes the patient to fall or to be otherwise unable to use the leg. Dislocation is followed by traumatic effusion into the knee joint and this further contributes to the weakening of the ligamentus reinforcement of the joint. The patient usually succeeds in reducing the dislocated patella himself except in those cases in which the bone takes a complete half-turn so that its outer edge rests against the anterior surface of the femur. In this case, the patella remains dislocated. The diagnosis of the condition is easy either from the actual findings at examination, if reduction has not been accomplished, or from the history which gives the characteristic mechanism by which dislocation occurs.

Treatment.—The treatment of the habitual dislocation of the patella by retaining braces or splints is usually insufficient unless such apparatus also greatly restricts the mobility of the knee joint; in this case, their practical value becomes very questionable.

When the dislocation occurs repeatedly, operative procedures are indicated. The method of Ali Krogius is an incision extending from above the patella downward to a few inches in front of the outer edge of the ligamentum patellæ. A bridge-shaped flap is formed on the inner side of the patella connecting with the tendinous expansion of the vastus externus and the fibrous capsule below and with the muscle and fascia above. This flap, left attached to its base both above and below, is now drawn across and sutured to the outer side of the patella.

Whitlock's method consists in a large horeshoe flap incision of skin and fascia which has its base in front and its apex backward toward the inner hamstrings. Then the ligamentum patellæ is exposed and divided by a blunt dissector in longitudinal direction. Into this slit in the patellar tendon the tendons of sartorious and gracilis muscles are threaded and sutured with chromic catgut.

Goldthwaite's Method.—A longitudinal incision is made 4 or 5 in. long extending from just below the tubercle tibia upward. Through this incision the patellar tendon is exposed and is split longitudinally in two equal halves throughout its entire length. The outer half is cut from its attachment below, passed under the inner half and attached to the periosteum together with the expansions of the tendon of the sartorius muscle at the inner side of the anterior surface of the tibia. If desirable, the patellar tendon may also be shortened. The wound is tightly closed and the leg is fixed in a plaster-of-paris bandage from the ankle to the groin.

Albee's Method.—The method of Albee consists in a semi-lunar skin incision made at the outer border of the patella sufficiently long to make the tibial tubercle accessible. With a broad, thin osteotome, the external condyle is penetrated, making a bone incision from 1½ to 2 in. long, and situated about ½ to ¾ in. below its anterior articulated surface and nearly in line with the long axis of the femur. From this bone incision, the anterior surface of the external condyle is elevated off to a plane above the internal condyle by producing a greenstick fracture of the external condyle reaching nearly to the intercondyloid groove. This creates a bone gap which is filled out by a transplant taken from the crest of the tibia through the lower portion of the original skin incision, cutting it out with a motor saw. The graft is drilled in one or two places so that bone pegs may be inserted which hold it securely in the bone gap of the external condyle. As advantages of this method Albee mentions the fact that no joint cartilage is sacrificed and that there is a minimum of injury produced at the time of operation which lessens both the danger of limitation of motion and that of formation of adhesions.

The writer has used the simple method of excising a crescent-shaped piece from the inner portion of the capsular ligament and suturing the edges together in a case of a girl 16 years old with habitual dislocation of the patella. No recurrence of the dislocation has been observed during the 18 months of observation which have since elapsed.

SPONTANEOUS DISLOCATION OF THE KNEE

The difference between spontaneous and habitual dislocation of the knee centers in the fact that habitual dislocation occurs from direct or indirect external forces even though they be of very slight intensity. In spontaneous dislocation the displacement is entirely subordinated to the will of the patient and can be produced as well as released at pleasure. Such voluntary dislocations have been observed in the humerus, the hip, the sternoclavicular joint, and in other articulations. Julius Wolff described a case of voluntary dislocation of the knee and another one is reported by Joachimsthal. In the latter instance, the dislocation could be produced actively when the foot was in equinus position and when the weight of the body was thrust forward. The flexion of the knee produced by contracture of the tendon of Achilles immobilized the lower end of the femur, and the forward thrust of the body caused the tibia to be displaced forward with an audible click. The patient experienced considerable discomfort when the knee was subluxated. By rotatory movement of the thigh he could reduce the dislocation.

GENU VARUM AND VALGUM

Synonyms.—Bow-leg and knock-knee.

The most common cause of knock-knee as well as bow-leg deformity is rickets. The deformity becomes established very soon after the first attempt at walking is made, which, as a rule, is somewhat delayed, sometimes until the age of 18 months or even later. In many cases, however, the deformity appears before the static functions have been established although it naturally increases very rapidly under the influence of static function.

In the knock-knee or genu valgum, the bend appears simply as an exaggeration of the physiological valgity of the knee. Anatomically, there are two distinct seats of the deformity; one is the lower end of the femur, and the other the upper end of the tibia. In the *x*-ray picture, one finds either a distinct kinking of the bone at the places mentioned or a rather arcuar deformity which reaches its apex at the side of the lower end of the femur or the upper end of the tibia. The deformity is recognizable at a glance, the patient walking with his knees touching and, from step to step, encircling each other as it were, so that one knee always seems to be in front of the other. There is more or less distance between the malleoli of the ankles. Valgus deformity of the ankle is a very common complication of knock-knee deformity, and there is definite mechanical connection between the knock-ankle and the knock-knee. The opinion has been ventured that a knock-ankle is in many cases responsible for the knock-knee deformity. This much is certain—that both deformities develop in frequent association upon a rachitic basis.

When the lower end of the femur takes part in the deformity, one finds on flexing the knees that the inner condyle of the femur reaches farther downward than the outer so that the joint line slants from the outward and upward to downward and inward. When the tibia alone is the seat of the deformity the slanting position of the condyles does, of course, not occur. Torsion of the tibia outward is very common especially in cases complicated by rachitic knock-ankles.

In severe cases, the patient walks with his knee flexed. This is partly due to the valgus position of the ankle associated with shortness of the tendo Achillis, which necessitates flexion of the knee in order to bring the sole flat to the ground; but the weightier reason for this attitude is the fact that in flexion of the knee the valgus deformity becomes masked so that the knee apparently becomes much straighter than in full extension. Part of this is due to the fact that beginning flexion of the knee is always accompanied by inward rotation of the tibia. Inasmuch as the tibia in these cases is unduly outwardly rotated, this maneuver provides a better alignment between tibia and femur and is in fact very often adopted to facilitate locomotion.

The genu varum deformity occurs also most frequently in the wake of rickets. In this deformity the bend is not as distinctly localized as it is in the genu valgum. It is rather common that the entire femur takes part in the bend. But here, too, it is the lower end of the femur and the upper end of the tibia which show most of the curving. In addition to this, kinking of the upper end of the femur and lower end of the tibia is often observed, and especially are noted the deformities of the lower tibial region which in some cases are the seat of most extreme deformities. The ankles place themselves in valgus position. In cases where the bend extends over the tibia this position is a compensatory one, as the ankle joint line then slants from inward-upward to outward and downward. When a child stands, the picture of the deformity becomes very evident. There is the outward rotation and the bowing of the upper end of the femur incident to the accompanying rachitic coxa vara, there is the wide spread between the knees, and there is the sharp kinking of the upper and lower ends of the tibia, and, finally, the compensatory valgus position. It must be noted that these children walk better than the patients with genu valgum. Consequently, they do not resort to the maneuver of flexing the knee to facilitate balance, but walk with their knees straight. There is, however, some difficulty in balance owing not only to the deformity of the knee but more so to the deformity existing in the hips (coxa vara) which produces a peculiar waddle not unlike that seen in double congenital dislocation of the hip. Frequently there is also a pendulous abdomen and an increased lordosis of the lumbar spine due to the higher inclination of the pelvis. All these factors contribute to produce a gait strikingly similar to that of congenital hip dislocation.

Prognosis.—With the conclusion of the florid stage of rickets and the return to a normal state of the chemical constitution of the bone, a natural halt is effected against further progress of the deformity. In the course of further growth gradual straightening of the limbs occurs under the influence of the law of functional adaptation of bone structure to its mechanical requirements (Julius Wolff). In young individuals this faculty of functional adaptation is considerably greater than in the adolescent period. Consequently, in the years following the formation of deformity, a gradual disappearance or diminution of the deformity can be observed, and many of the milder cases undoubtedly go on in this way to automatic and spontaneous cure. It must not be forgotten, however, that this faculty of self-correction moves in rather narrow limits and that beyond certain degrees a spontaneous cure of the deformity should not be expected. In the light of the fact that most of these deformities develop and that all of them increase after the static functions of the limbs have been assumed by standing or walking, while, on the other hand, the first signs of florid rickets appear much earlier in life, considerable attention must be given to the prevention of this deformity.

Children with rickets should not be encouraged to stand or walk, since their refusal to do so is well founded by reasons of chemical and histological anomalies of bone formation. Anti-rachitic treatment, massage of the muscles, and application of ample rest will do much in preventing the establishment of rachitic deformities of knock-knees or bow-legs. When the children insist on standing up and the bowing has not gone beyond moderate limits, contentive dressings with plaster-of-paris apparatus applied to the limbs effectively prevent further progress of the deformity. When the bones are still soft and pliable, which is the case usually up to the end of the second year or somewhat later, actual correction of the deformity may be accomplished by gradual correction in a series of casts. These casts should be changed about every month, each succeeding one giving the limb a little more additional correction. In the course of four to six months one will then note that moderate knock-knee or bow-leg deformities yield nicely to this type of conservative treatment. When the desired correction is obtained, the plaster-of-paris cast may be omitted but it is then usually necessary to apply protective splints which should be worn well into the second or third year of life because of the tendency of recurrence of the deformity which is ever present as long as weight is being borne and the bones have not become thoroughly hardened. After the removal of the casts systematic massage of the weak and flabby muscles of the extremity must be added to the treatment.

Cases in which the deformity has reached a higher degree or cases in which the deformity has been allowed to go on to an age at which the bones are already hardened or eburnated, will not yield to conservative means and are therefore subjects for operative procedures. The most widely adopted surgical procedure is the osteotomy. Langenbeck was the first to perform subcutaneous osteotomy in 1854 using a small incision of 1 to 2 cm. length through the skin and soft parts. Adams and Moore and recently Hoffman availed themselves of a pointed saw in place of an osteotome. When doing an osteotomy of the knee for knock-knee deformity, it is not necessary to divide the entire bone with the osteotome but one-third to one-half of the bone divided will allow the rest to be broken by manual force. In the so-called circumference osteotomy of Reiner, a special instrument is used by which the cortical substance of the bone is divided over one-third to one-half of its periphery and then the bone is broken by manual force. In either knock-knee or bow-leg deformity, the place for the osteotomy of the femur is above and near the lower femoral epiphysis. The osteotomy through the epiphysis itself is not to be recommended because of the probability of causing grave disturbances of growth from injuries to the epiphyseal disc. The place for osteotomy of the upper end of the tibia is below the upper epiphyseal line at a point where the kinking of the bone is most prominent. It is not advisable to do multiple osteotomies of one bone or even one extremity. This entails an unnecessary prolongation of the operation and involves definite risks. Among these, the danger of fat embolism, though remote, should not be overlooked. It is not so likely to occur in comparatively healthy bones of the rickets as it is in the deformities existing in infantile paralysis or following inflammatory conditions of the knee.

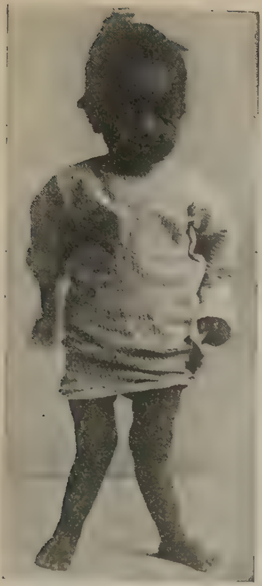

FIG. 241.—Rachitic genu valgum before osteotomy.

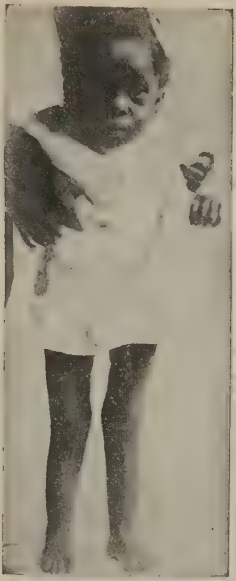

FIG. 242.—Rachitic genu valgum after osteotomy.

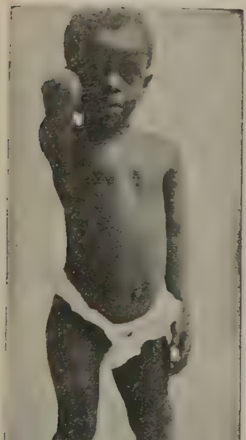

FIG. 243.—Bowing of tibia before osteotomy.

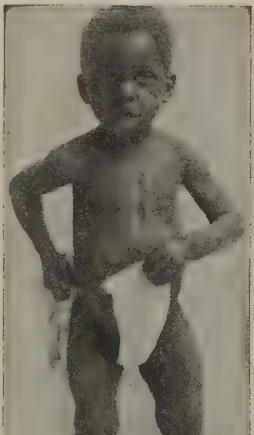

FIG. 244.—Bowing of tibia after osteotomy.

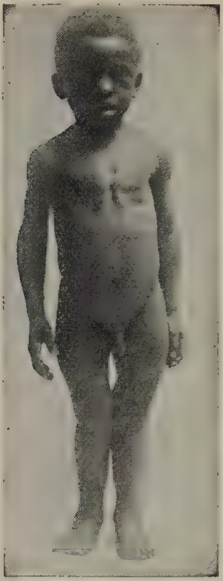

FIG. 245.—Bowing of tibia after osteotomy.

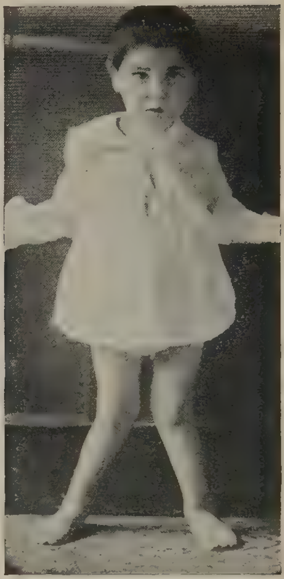

FIG. 246.—Rachitic genu valgum before osteotomy.

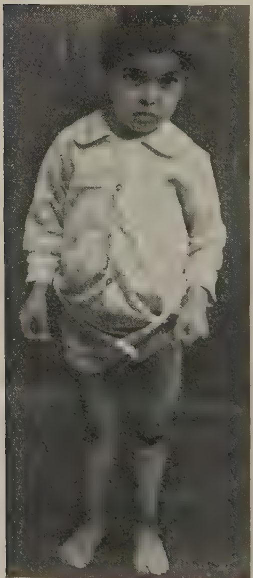

FIG. 247.—Rachitic genu valgum after osteotomy.

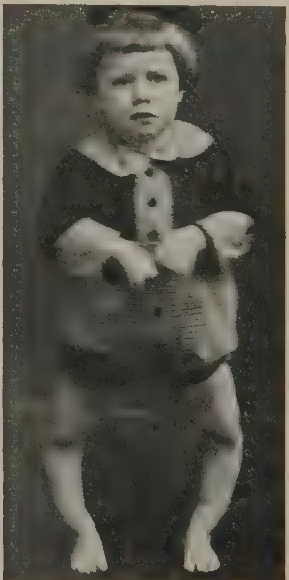

FIG. 248.—Rachitic genu varum.

Osteoclasis or the bloodless breaking of the bone is a procedure which has found many adherents. Dr. Blanchard of Chicago who used the Grattan osteoclast had probably the largest experience on record with this method. He emphasized in favor of this instrument that its great weight gives it an advantage over the osteoclasis of either Lorenz or Rizzoli. The operation of osteoclasis can be performed with great rapidity, in fact within a few seconds, and due to this rapid osteoclasis, the soft tissues which are pressed against during the operation quickly spring back into their normal condition so that circulation is immediately restored and no definite injury is inflicted. However, objections have been raised to this procedure by De Forest Willard and J. K. Young on the grounds that the osteoclasis lacks precision, that it splinters the bone, and entails the danger of separation of the epiphyses, complications which do not occur in the open operation of osteotomy. Consequently, a number of orthopedists limit the osteoclasis to the shaft of the tibia. Considering the simplicity of the open operation which can be performed from a very small incision through the skin and which obviously better preserves the soft tissue, there can be no serious objection to the use of osteotomy as a routine method in the correction of bow-leg and knock-knee deformities.

Following the operation the position must be maintained by plaster-of-paris dressings reaching from the toes to the groin and holding the limb in over-correction. The children should be encouraged to bear weight at the earliest possible moment since this has a decided influence upon the firm union of the fragments. That the danger of non-union in these cases is not so remote as might be generally supposed can be testified by the writer who in two cases of osteotomy for deformities of the tibia has seen non-union develop. The cast should remain for two or three months at the most and it should then be replaced by a brace or splint and massage and exercises should be added to the treatment. Walking or standing should be encouraged as early as four weeks after the operation.

Congenital Genu Varum.—A case of congenital genu varum is described by Cramer, and others are described by Joachimsthal, by Ehrlich and others. In a number of these cases the principal pathological finding consisted in a bony attachment coming from the lower end of the femoral epiphysis at its median portion. This was considered to be an anomalous and rudimentary tibia which had become synostotic to the femur. A case of Roskoschny is described as follows: The patient was 39 years old when seen, and he as well as his son 3½ years old showed the knee joints in valgus position caused by a completely ioslated internal condyle of the femur of considerable size which articulated with the tibia. In the case of Cramer, the tibia was well developed but the femur showed the bony attachment which was described above. Familial tendencies of these cases speak for the endogenous nature of the deformity. In addition to this, other deformity coexisted in some of these cases, such as amniotic scars and deformities of the elbow joints. In another number of cases, however, no concomitant deformities were found.

TRAUMATIC DEFORMITIES OF THE KNEE JOINT

Sprain of the Lateral Ligament of the Knee.—The strain of the internal lateral ligament of the knee produces well-localized and characteristic symptoms which make the diagnosis easy. The pain is strictly located at the side of the internal lateral ligament and a distinct tenderness exists in the joint fissure at the site of the ligament. This tenderness is distinctly posterior to that existing in injuries to the internal semi-lunar cartilage. Strain of the internal lateral ligament of the knee is caused by direct or indirect violence which results in an attempt at abduction of the leg, and which is checked by the internal lateral ligament. Strain of this structure may further be caused by static influences existing either in the knee joint or in the ankle joint. In genu valgum, the strain upon the internal lateral ligament of the knee is considerably increased. A similar strain is produced upon the internal lateral ligament when the foot assumes valgus position in the ankle joint as is the case in the static pronated foot. Hence, the frequency of pain in the knee in cases of static valgus. Acute violence of severer degree may produce a complete rupture of the internal lateral ligament with penetration into the synovial lining of the knee joint and a resulting intra-articular hemorrhage. Finally, since the internal semilunar cartilage is closely woven into the reinforcing ligaments at the inner side of the knee, a rupture of the internal lateral ligament may also contribute to the displacement of the semilunar cartilage.

Treatment.—The treatment consists in rest, bandaging, and later massage of the knee joint. By applying an inner wedge to the heel and sole of the shoe the strain upon the knee joint from a valgus ankle may be effectually relieved. Severe ruptures of the internal lateral ligament demand immobilization of the joint preferably in plaster cast for a period of from two to six weeks. After this, the patient should wear a splint which while allowing flexion and extension prevents lateral motion of the knee.

Displacement of the Semilunar Cartilage.—The semilunar cartilages of the knee joint represent two crescent-shaped structures of fibro-cartilaginous texture placed upon the internal and external condyles of the tibia and articulating with the cartilaginous surfaces of the tibial condyles below and the femoral condyles above. The cartilages are covered by endothelial layers which are duplications of the synovial membrane. The inner and outer semilunar cartilages differ both in shape and relations to the surrounding tissue. The inner cartilage is C-shaped. Its anterior horn is fastened in front and behind the tibial spine. In its outer circumference it enters into close relation with the ligamentous apparatus at the inner side of the knee, being closely attached to the structures of the capsular reinforcement. The outer semilunar cartilage on the other hand is more circular in shape. The attachment of its horns in front and behind the tibial spine are much closer together and are within the distance lying between the anterior and posterior horns of the inner semilunar cartilage. Consequently the inner cartilage is much less movable than the outer and cannot evade injuries as readily as the outer cartilage which by virtue of its more complete circular form and its less intimate relations with the surrounding capsular tissue enjoys more freedom of motion. This is the main reason why injuries to the inner semilunar cartilage are so much more frequent than those to the outer cartilage. The injuries to the internal semilunar cartilage occur in a rather typical way. Usually the knee is in flexion, the thigh somewhat inwardly rotated while the injury tries to force the leg in outward rotation and abduction. During this abduction the inner side of the knee joint is opened and the cartilage is forced out. When the abduction ceases a moment later, the median and anterior portions of the internal semilunar cartilage are caught. The result is a distinct tear of the structure. Sir Robert Jones states that of 117 cases on which he had operated a number of years ago, 53 cartilages were found torn from their anterior attachment; 16 were split longitudinally; eight were torn from the capsule while their horns remained attached; in seven there was a displacement of the posterior horns; 12 were fractured transversely opposite the internal lateral ligament.

Symptoms.—At the moment of the injury the patient feels a sharp pain which often causes him to fall to the ground. The knee cannot be fully extended. A distinct pain appears which is located at the site of the internal semilunar cartilage immediately in front of the internal lateral ligament. Very shortly afterwards an effusion occurs into the joint which may persist for several days. The patient who is still unable to fully extend the knee may now be able to bear weight on it, but always does so with limp and considerable discomfort. This distinct lockage of the knee joint against full extension is a characteristic symptom which indicates a displacement of the cartilage with mechanical interference of the free motion of the joint. Sometimes the patient is able immediately after the injury to manipulate his cartilage into place so that full extension again is possible. More often, however, he cannot do so and the disability forces the patient to seek medical attention.

Treatment.—The immediate indication is the replacement of the semilunar cartilage by manipulation. If necessary under the influence of an anesthetic, the joint must be manipulated in a systematic way so as to accomplish a reposition of the dislodged cartilage. The maneuver is flexion of the knee, abduction of the leg upon the thigh, followed by inward rotation of the leg, and, finally, forcible extension. Sir Robert Jones, whose experience of these cases excels that of any other observer, as he has seen upward of 2000 cases of this kind, stabilizes the thigh of the patient with one hand while with the other he pulls the leg downward and outward. He then directs the patient to kick forcibly, and in this attempt the semilunar cartilage is replaced. Almost without exception, the patient is fully aware when replacement occurs and will so advise the physician. Following a successful reduction the limb must be bandaged and the bandage must remain until all traces of the secondary effusion into the joint have disappeared. Full extension of the knee is necessary to prove successful reduction and it is entirely useless to cease with manipulations before this has been accomplished. Following the successful reduction and after the bandage has been removed, a hinged knee-iron may be worn for some time which protects the knee against lateral abduction.

Recurrent dislocations of the semilunar cartilage are very common. It must be remembered that with each attack a traumatic effusion into the joint occurs which gradually renders the joint weaker and more subject to similar attacks in the future. Cases in which manipulative reduction of the displaced cartilage has failed, or cases in which repeated attacks occur, especially in boys or young men, cases which concern structural iron workers or persons working under conditions in which a fall might bring on severe consequences, should be operated on.

Operative Procedures.—The operation upon the semilunar cartilage involves the opening of the knee joint and is therefore a procedure which is not without risk. This imposes upon the surgeon the necessity of the greatest possible care and skill as well as the most scrupulous observation of aseptic technique. The knee should be carefully prepared for the operation by thorough cleansing and by covering it with sterile bandages for a period of 48 hours. The best approach to the internal semilunar cartilage is that advocated by Sir Robert Jones. An incision is made 1 to 1½ in. in length over the upper border of the internal head of the tibia parallel to the semilunar cartilage and slightly sloping downward and inward. From this incision the cartilage can be reached easily and can be removed with curved scissors after it has been drawn forth with a hook. Under no circumstances should the gloved finger itself be introduced into the interior of the joint. It is better to remove the cartilage than to try suturing. The latter procedure is not to be recommended.

A report on 262 operations by D'Arcy Power from St. Bartholomew Hospital in London (1911) covers a period of 17 years. The results of these operations were brought out by 89 replies received from patients who had been operated on. No return of symptoms was stated in 73 cases while 16 had recurrences. The knee was of normal strength in 50 cases but it was weaker in six. The movement was perfect in 77 cases and was impaired in 12. The shape of the knee was normal in 68 cases and was slightly changed in 21. The knee was painless in 43 cases, occasionally painful in 35, and continuously so in 11 cases. The ratio of recurrence, therefore, according to this series was only 2:9, which demonstrates that this operation is of considerable value.

It is the advice of Sir Robert Jones to let the patient up as early as three or four days after operation, but he should not bear weight upon the leg until a week following the operation and then only with the knee properly bandaged or supplied with a posterior splint. Ten days after the operation one should begin with massage and should continue with it until the strength of the knee is fully recovered and the girth of the thigh has returned to normal dimensions. Sir Robert Jones' cases were able to return to work as early as five weeks after the operations and some of his athlete patients were able to take up football work without recurrence of symptoms.

In cases in which the semilunar cartilage cannot strictly be located at the inner side of the knee and in which a displacement toward the interior of the joint is suspected, the approach should be that of the transpatellar route. This approach which will be described in detail in the section on "Osteochondritis Dissecans of the Knee" gives an excellent view of the entire anterior portion of the knee joint to and beyond the crucial ligaments.

Differential Diagnosis.—A number of conditions may simulate the picture of the displacement of the internal semilunar cartilage. The most important of these are the impingements by free bodies of the knee or by synovial fringes.

The free bodies of the knee joint may be divided into three groups: (1) Those of fibrous character which are due to organization of fibrinous exudates, some of which are designated as rice bodies. While these bodies cause impingements, they do not produce the severe symptoms seen in semilunar cartilage displacement because the impingement is usually of short duration and is comparatively easily released by motion of the joint. (2) Loose bodies of cartilaginous or osseous orign may be produced by injuries to the knee joint which cause the breaking off of bony spurs such as may exist in different stages of arthritis deformans. These are also capable of producing symptoms of impingement which are also of milder character and are indefinite in location. (3) Impingement due to hypertrophied fat-fringes also causes mechanical disturbances of the knee. These fringes are usually the outgrowth from the retropatellar fat pad which protrudes into the interior of the knee joint. These patients complain of pain upon extension of the knee and have difficulties in getting up from a chair or in walking up or down stairs. Secondary effusion in the knee joint follows. For the relief of this latter condition, a hinged brace is advised by Sir Robert Jones. This brace does not permit of full extension of the knee, stopping within 30° of full extension. In addition to this, a heel pad should be worn under the heel and inside the shoe raising the heel about ½ in. and forcing the patient to walk with his knees slightly flexed.

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Rupture of Crucial Ligaments.—The rupture of the crucial ligaments is one of the severest injuries to the knee joint. The force required is usually of a sufficient intensity to cause an actual dislocation of the knee. Owing to the very distinct function of the crucial ligaments as stabilizers of the knee joint, the diagnosis, as a rule, offers no great difficulties. The functional disturbances following this injury may be readily understood if one keeps in mind the specific checking mechanism of these ligaments. The anterior crucial ligament checks the forward motion of the head of the tibia, and the posterior ligament the backward motion. In addition to this, the anterior crucial ligament becomes tense in extension, and the posterior in flexion of the knee joint. Rupture of the anterior crucial ligament therefore permits a forward movement of the head of the tibia. When the patient puts his foot to the floor he may actively force the tibia forward sometimes with an audible click. In cases of rupture of the posterior crucial ligament the head of the tibia can be displaced backward. When walking this displacement occurs at the end of the step just as the patient is about to lift the foot off the floor. In cases of rupture of both ligaments, the knee is distinctly flail and the patient is unable to bear weight on the knee or to walk.

In 14 cases reported by Hey Groves, the anterior ligament alone was torn in 10 cases, the posterior in two cases, and both ligaments in two cases. In none of his cases was there any *x*-ray evidence of any part of bone having become avulsed from tibia or femur. In most instances, the ligaments were ruptured at the femoral attachment.

Owing to the severity of the injury, the lateral reinforcing apparatus of the knee joint is usually involved also. The immediate result of the injury is a most marked effusion of blood into the joint and the abnormal mobility which has already been mentioned.

Treatment.—A number of cases show good recovery upon prolonged immobilization in plaster of paris. Sir Robert Jones advises absolute immobilization for a period of from four to six months, preferably in a plaster-of-paris cast. After this time a splint should be worn during the day to be removed at night. Only when the abnormal mobility of the joint has disappeared the splint may be discarded and substituted by a short jointed splint, the range of motion of which should be carefully graded and slowly increased.

In cases of long standing, conservative methods often prove insufficient. The first one to operate upon the ruptured crucial ligaments was Mayo Robson who employed suture. It appears however that suture is extremely difficult because of the destruction and attrition of the ligaments due to the injury. In 1913 Goetjes reported 30 cases, 12 of which were operated. A fragment of bone was removed in five cases and repair of the ligament was effected in a like number of cases. Hey Groves operated his first case in 1917 and in 1920 reported on 14 cases operated by this method. He uses a large U-shaped incision made in front of the knee and crossing the tibial tubercle. The tubercle is chiselled off ¾ in. and an incision is made through capsule and synovia. The tubercule with the patellar tendon and the patella is now turned upward. Then the knee is flexed, the ilio-tibial band is defined by prolonging the outer end of the incision and from this band a flap 8 by 1½ in. is separated with its base distally at the original attachment of the band to the head of the tibia. Then the condyle of the femur is drilled from outward inward so that the inner end of the hole is placed far back on the outer aspect of the intercondyloid notch. Next, the front part of the tibia is also drilled from outward inward, the outlet being in front of the tibial spine. The strip of fascia is then passed through the external orifice in the femur, then out through the internal orifice, then obliquely through the internal orifice of the tibia, and out through the external orifice of the tibia. From here the terminal part of the band is turned upward to supplant the internal lateral ligament.

The posterior crucial ligament is formed as follows: The same incision is made only this time the inner line of the U-shaped incision is lengthened upward 3 in. Then the tendons of the gracilis and semitendinosus are defined and isolated by cutting them off 6 in. proximally, leaving again the distal ends of the tendons attached. This slope of tendon is then brought forward into the joint. Drill-holes are laid through the internal condyle of the femur as far forward as possible coming out at the inner aspect of the intercondyloid notch. The tendons are pulled through this hole by means of ribbon gauze wrapped around them, the knee is extended and then the free ends of the tendon are turned down and are attached to the inner aspect of the inner tuberosity of the tibia.

The after treatment consists in the application of a posterior splint which is followed two weeks later by daily massage and exercises.

A similar ingenious method of reconstruction of the crucial ligaments has been devised by Putti who uses fascia material for the formation of the artificial ligaments.

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OSTEOCHONDRITIS DISSECANS

It is only occasionally that this condition is observed in younger individuals. It is essentially an affection of the adolescent and middle age, but a short description of the condition is not out of place because it is a factor in the differential diagnosis of the different traumatic lesions of the knee joint. This condition is characterized by the distinct and localized involvement into a process of necrosis of the outer edge of the inner condyle of the femur. In the *x*-ray picture, this necrotic area presents itself as a blurred spot in which the texture of the bone has been lost. Later, a deposit of lime salts occurs which gives outline and distinctness to the *x*-ray picture simulating very much the condition seen in osteochondritis deformans of the hip or Legg-Calvé-Perthes' Disease; but while in the latter disease the fragmentation of the epiphyseal head is followed by restitution, in osteochondritis dissecans of the knee the necrotic bone becomes ultimately detached from its matrix and falls into the joint where it may form a loose body and thereby a mechanical obstacle to the joint motion.

In fact, it is this factor which is most prominent among the clinical symptoms and which prompts the patient to seek surgical care.

Almost all cases are due to traumatism. The mechanism of the injury is usually a blow or fall upon the knee joint in a position of extreme flexion. In this position the knee joint is exposed in its anterior aspect and a blow to the joint will transmit itself through the patella directly upon the outer surface of the inner condyle of the femur near the intercondyloid notch. The affliction may therefore be considered as a sort of aseptic necrosis due to trauma and followed by absorption of bone in a manner similar to what is seen in post-traumatic kyphosis of the bodies of the vertebræ. When these knees are inspected one will find a bluish discoloration of the cartilage at the place of the necrosis. Underneath the cartilage the bone is soft and may easily be scooped out with a curette or the necrotic piece of bone may be found loosely attached to its matrix. When the body has become separated and has dropped into the joint, the defect is soon filled out by overgrowing cartilage tissue so that at operation no defect in the cartilage lining is seen. Freiberg has made a thorough investigation into the histology of the necrotic pieces. Sections of the free bodies showed that they are composed of a central area containing lime salts in small amounts and a small amount of yellowish material resembling blood pigment. Next to the central area there was a homogeneous and hyaline area containing groups of cartilage cells. The examination of the bodies by Brackett proved them to consist mainly of cartilage with newly formed bone in the center. Microscopically, the cartilage cells appeared in small clusters surrounded by a homogeneous, hyaline matrix which towards the periphery became more dense and more fibrous in character.

The clinical symptoms have their main expression in the limitation of motion caused by the impingement of the fragments. When the fragment recedes into the interior of the joint, it may not interfere with motion but if interposed between the condyles of the femur, motion will be impaired and lockage occurs. In the cases mentioned by Brackett, there were no other pathological findings outside of the area of detachment excepting in one case in which changes indicating arthritis deformans existed.

Treatment.—The treatment is surgical. The operation used by Brackett is the transpatellar approach to the knee joint. The incision is made from the middle of the anterior surface of the thigh downward corresponding to the run of the fibers of the rectus femoris muscle and therefore slightly inclined downward and inward. Running over the patella it divides this bone into a somewhat larger outer and smaller inner half while over the ligamentum patella it again has a slightly outward swing following the direction of this structure. The fibers of the muscle are divided bluntly, the patella is sawed through, and after division of the fibers of the ligamentum patella, the halves of the bone are drawn apart and the knee joint flexed bringing the anterior portion of the joint into full view.

The writer has encountered little difficulty in locating and removing the body when following this method except in one case in which the body had become attached to the head of the tibia, being matted so completely into the surrounding cartilage that its detection was not easy. At the removal of the body the wound is closed and the limb immobilized. The patient may be allowed to walk in two or three weeks in a well-molded posterior splint. Massage should begin early and active motion should be added after four weeks when the union of the patella is sufficiently firm.

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OSGOOD-SCHLATTER'S DISEASE

Synonym.—Apophysitis Tibæ.

The honors of having called attention to a disturbance at the tibial apophysis as a distinct clinical identity are being shared by Osgood and by Schlatter, both of whom have published their articles in 1903.

The trouble consists in a swelling and tenderness over the tibial tubercle expecially noticed upon extension of the knee. It is an affliction of the adolescent age, occurring most often between the twelfth and fifteenth year of life, almost exclusively in boys, although cases have been observed to occur later in life. In the *x*-ray picture, distinct changes are seen in the beak-shaped process which joins the epiphysis of the tibia and which is known as the apophysis of the tibial tubercle. Although distinct changes are seen in radiograph, the picture is by no means uniform. In some cases, there appears a separation between the tibia and the tongue-shaped process. In others there is a distinct fragmentation in several pieces or a clean fracture through the base of the process. Regarding the etiology of the condition, the opinions appeared to be divided between the traumatic and inflammatory nature of the disease. Schlatter is of the opinion that the condition is a fracture of the beak-shaped process of the upper tibial epiphysis. This opinion has been emphatically opposed by Alsberg and Blencke who believed the disease to be of inflammatory origin and correspondingly speak of an apophysitis of the tibia. Hoglund has reported a series of 20 cases, the study of which leads him to follow the opinion of Schlatter, and to adopt the traumatic theory.

The question as to the etiology of this disease is important from the viewpoint of treatment. As a mere traumatic condition the simple means of immobilization would appear sufficient, whereas in the case of an osteitis operative interference is justified. Such operative interference was carried out by Lenander who operated on several patients, but Hoglund remarks that signs of inflammation were not found at microscopical examination.

Another theory is advanced by Matsuoka who sees an anomaly in the ossification process of the tuberosity of the tibia, as the cause of the disease. On the other hand, Alsberg in describing his operative finding mentions the fact that granulation tissue with round-cell infiltration was found histologically, but the specimens removed were always sterile. Unfortunately, the histological findings which alone would be able to throw light upon the matter of the etiology of the disease are very scant and incomplete.

It seems that immobilization in plaster of paris for two or three months controls the symptoms in most of the cases. No doubt a number of these cases are merely traumatic in character while others have an inflammatory background. Cases showing clean fracture line in the *x*-ray picture can hardly be explained in any other way but as traumatic. Other cases show a fragmentation of the apophysis with irregular outline of the shadows, but clinically a thickening and tenderness of the periosteum. In these cases, an inflammatory background may be assumed. The disease seems to be more often unilateral than bilateral. The bilateral cases are hard to explain on a traumatic basis, but again in most of the cases the trouble does not occur simultaneously on both knees but at intervals. That the disease has been known before the publications of Osgood-Schlatter can be seen in the work of Aboukir who wrote a thesis on the apophyseal osteitis of the tibia in 1899.

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FIBROUS ANKYLOSIS OF THE KNEE

For practical reasons it is opportune to consider ankylosis of the knee collectively although it only represents a symptom which may arise from conditions which differ markedly in their etiological background. Ankylosis of the knee occurs when the pathological changes result in obliteration of the normal joint surfaces either by total or partial destruction of the cartilage. In this event, granulations penetrate into the joint from the denuded surfaces of bone and produce at first fibrous and later, eventually, bony bridges which are mechanical obstacles to free motion. In the vast majority of cases, these changes are the result of a definite infection which is responsible for the destruction of the joint surfaces. From a practical standpoint, a clinical distinction made by H. O. Thomas is of great importance. He distinguishes between sound and unsound ankylosis. A sound ankylosis is one in which the inflammatory condition has subsided. An unsound ankylosis is one in which the inflammatory condition is still present, though perhaps dormant temporarily. In practice, the distinction between these two types is by no means easy; yet it is necessary to make such a decision as closely as possible because of the entirely different reaction to treatment shown by the one or the other type.

Inasmuch as the formation of adhesions is proportionate to the intensity of infection and the duration of the inflammatory condition in the knee, it may be stated in general that the best prevention of the formation of adhesions lies in measures which control the inflammatory condition itself. Immobilizing, therefore, not only mitigates the actual inflammation but indirectly restricts the later formation of adhesions. It is erroneous to believe that immobilization itself produces adhesions. On the contrary, inasmuch as it limits infection, it also lessens the probability of the formation of adhesions or at least diminishes their intensity. Fibrous adhesions within the knee joint may follow any kind of infection. In mild cases, the fibrous ankylosis may be influenced by application of heat, passive congestion, and massage inasmuch as a gradual giving of fibrous adhesions will occur under these measures especially if they are duly alternated with immobilizing measures such as splints or braces. The forcible manipulation of the knee joint or the breaking of adhesions by force, the so-called brisement force, is to be condemned on general principles. It never adds a particle to the range of motion of this joint, it is regularly followed by severe reaction, very often by a recrudescence of the inflammatory conditions, with the result that the adhesions recur only in greater numbers and with increased intensity. It may be argued that the position of the knee may be improved by forcible manipulation in case of a flexion contracture. On the other hand, the results of gradual correction of contracted knees by plaster-of-paris casts without use of force are so encouraging that the value of forcible manipulations is very doubtful even as a means of correcting position only.

There is another danger involved in the brisement force of the knee and that is the fracture of the lower end of the femur which occurs especially easily in joints which have not borne weight for a certain length of time and in which there is considerable atrophy of bone.

The degeneration of the bone marrow into a fatty structure with very little cohesion also opens up the possibilities of fat embolism following forcible manipulation of the knee, a complication which should always be taken into consideration. Cases of fat embolism have been reported frequently, one of the most instructive series being that of Aberle. The symptoms usually appear within a few minutes to a few hours following operation. The fat embolism is always a grave complication and the mortality is high. The principal symptoms are cyanosis, dyspnea in cases of fat embolism of the lungs, and convulsions and other cerebral symptoms in cases of cerebral emboli.

Sometimes forcible stretching of the knee is attended by injury to the popliteal nerve, especially the external popliteal, with resulting paralysis of this nerve. This branch is especially subject to injury by stretching because of its intimate connection with the head of the fibula to which it is firmly attached so that the full force of the stress is exerted upon this portion of the popliteal nerve. In some cases, convulsions have been observed as a result of a reflex action following the stretching of this nerve. In this event, the plaster-of paris cast should be speedily removed in order to eliminate the reflex irritation.

It has already been mentioned that in contrast to the forcible methods of stretching contracted knees, the methods of gradual stretching by series of corrective casts offer much better clinical results and involve much less danger to the patient The writer has treated a considerable series of cases of knee contractures due to extra- or intra-articular causes. It is well to apply the cast under anesthesia to relax the musculature of the knee and to give the joint the best possible position it can assume free from the influence of reflex contraction. In this state, corrective knee casts can be applied reaching from the toes to the groin. When application of force is studiously avoided one will find that the patients are quite comfortable afterwards and that it is not necessary to apply opiates.

Every two or three weeks the cast should be changed, adding first a little more correction until the correction of the knee has been completely accomplished. Then the cast may be exchanged for braces of caliper type with knee-caps to hold the knee down firmly and to counteract any tendency to contract. These braces should be worn day and night. At this time, also, it is appropriate to begin with massage and gentle motion, both active and passive, and to teach the patient gradually to stand and take steps. The latter procedure requires a good deal of care and patience especially in cases which have been bedridden for a number of years.

BONY ANKYLOSIS OF THE KNEE

The correction of bony ankylosis of the knee is naturally an operative problem. The flexion deformity can be corrected by osteotomy. Ollier's supracondylar osteotomy of the femur is the method which is most frequently used. An arcuar osteotomy has been used by Helferich and Codivilla. When operating at the site of the ankylosed knee a wedge-shaped osteotomy may be used in which the base of the wedge is laid toward the convexity of the deformity. The operative immobilization following osteotomy may vary according to the underlying condition but should always be sufficiently long to allow for complete bony union. The plaster cast is applied at the time of operation, and, as a rule, should be left for two to three months. At the same time, it is important for the formation of an early and sufficient callus that the function of the limb should be taken up as early as possible. Therefore, the patient should be encouraged to bear weight and to walk in the cast as soon as there is a definite callus formation and as soon as weight-bearing may be borne without pain. This may be the case in from four to six weeks after operation. In many cases it is advisable to apply a retaining splint or a brace for a longer period of time. This is necessary especially in those instances in which the ankylosis is the result of the inflammatory destruction and in which the bone vitality is necessarily much impaired. The failure of following up the immobilization period by a long period of splints or braces is responsible for the occurrence of secondary deformities after osteotomies of the knee joint.

Deformities Following Knee Resection.—Resection of the knee joint should not be applied to the juvenile or infantile age but should be reserved for adolescent or adult cases barring a few exceptions in which vital indications make the operation unavoidable at an earlier age. Kirmisson found among all the cases of knee resection in children which he had followed up not one which gave a satisfactory result. The results of late examinations of resections of the knee performed before the thirteenth year in the clinic of von Bruns brought out that only 27 per cent. of these knees remained straight; all others showed subsequent deformities of one or the other type.

The most common deformity which follows resection of the knee is that of flexion of the knee and of adduction of the leg so that a genu varum deformity results. In children it seems very difficult to prevent deformities following resection of the knee, mainly because it is hard to control the influence of epiphyseal growth. With the removal of the tibial head and its epiphyseal cartilage or the removal of the epiphyseal ends of the femur with more or less irregularity, it is clear that an unevenness of the growth of the extremity will ensue which must later lead to deformity. Many authors express the opinion that the only way to prevent these unfortunate deformities following resection of the knee is to refrain entirely from the practice of resection during the infantile and juvenile ages and to postpone such interference until the patients have become adolescents.

Arthroplasty.—Arthroplasty of the knee joint should not be carried out in patients under 15 or 16 years of age. Even then, the indications for this operation must be made with a great deal of judgment. Osteotomies and resections of the knee on one hand give excellent functional results if performed at proper age, while, on the other hand, the functional results following the arthroplasty of the knee are still uncertain. The knee joint is the least favorable for the performance of an arthroplasty. A number of methods have been devised for this procedure. Probably the most satisfactory results have been obtained by the method of V. Putti. This surgeon makes a U-shaped incision around the patella with the base of the flap pointing downward. After the flap is prepared and deflected downward, the vastus internus and externus are defined and separated from the rectus femoris muscle. After this is done, the quadriceps tendon is split longitudinally, both halves being freed at the upper and lower ends of the split by short transverse incisions. The patella is then turned down together with one-half of the split tendon. Then the articular ends of the knee are dissected from the anterior surface, and this is followed by the arthrolysis or the freeing of the condyles of the femur from the condyles of the tibia by means of specially shaped chisels which are driven into the synostotic bridge from in front and which divide the bone way back over the posterior surface. Another pair of specially shaped chisels is used for the shaping of the condylar surface of the tibia. When this is done correctly, joint surfaces similar to the normal are created.

There is then a suitable intercondyloid notch between the condyles of the femur and a corresponding eminence of the tibia, representing the tibial spine, between the condyles of the tibia. This arrangement is of great importance to the lateral stability of the newly formed knee joint.

As interposition material, Putti uses a flap taken from the fascia lata which is reached by lengthening the outer branch of the incision. This fascia flap is now interposed and carefully sutured to the posterior capsule as well as to the anterior structures of the knee. Putti lays great stress not only upon the proper shaping of the condyles of femur and tibia but also upon the smoothing and polishing of the joint surfaces using for the latter purpose a selection of round or curved files. Where the patella is adherent it must be freed from the anterior surface of the tibia by a chisel before it can be turned downward together with one-half of the quadriceps tendon. After the flap interposition is completed, the halves of the patellar tendon are reunited giving the tendon the proper length for free motion. The limb is now placed in the plaster-of-paris cast in which it remains until the aftertreatment consisting of massage, active and passive motion may begin, or, following Murphy's procedure, the first weeks after operation are spent in bed with an extension applied to the knee. When the aftertreatment begins, a removable splint is applied instead of a plaster-of-paris cast. In cases which are treated by extension, however, the aftertreatment begins within a week or two after operation and is carried out in bed. As in all cases of arthroplasty, the greatest amount of care and judgment is necessary to avoid pain and irritation and to subdue the traumatic reaction which follows the operation.

SYNOVITIS OF THE KNEE

Serous effusion into the knee joint is a symptom of reaction of the synovial membrane to irritative agents of either traumatic or inflammatory nature. The flooding of the knee joint with fluid follows so regularly the agencies of this type that it in itself cannot serve to identify any specific nature of infection. It is well known that chronic tuberculous disease of the joint bodies produces an effusion of synovial fluid prior to the time at which actual perforation of the tuberculous focus into the joint cavity takes place.

Chronic infectious arthritis of the knee joint is also associated with effusion of fluid which at first maintains a serous or sero-fibrinous character. The direct pyogenic infection of the knee joint which produces suppuration of the joint cavity is associated with symptoms so marked and striking that the presence of a pyarthrosis or pus formation in the joint will rarely be in doubt. All traumatic conditions about the knee joint in so far as they do not directly cause effusion of blood into the cavity are followed by a reactive synovitis which is primarily of serous or serofibrinous character. There remain, however, certain types of chronic effusion into the knee joint which cannot be associated with definite trauma or definite infection and the nature and pathogenesis of which are consequently more or less uncertain. Effusions into the joint not uncommonly follow injuries. This may be explained by organic changes which have taken place in the synovial membrane after the injury and which subsequently render the capsular sac so sensitive to stress and strain that it reacts with repeated effusion even to the everyday stresses of the static function of the knee.

A condition known as intermittent hydrops is occasionally observed in children and young individuals. There is often present a certain periodicity which is extremely difficult to explain. In cases of young girls sometimes a relation exists between the intermittent hydrops and the menstrual period. But intermittent hydrops is also seen occasionally in men at regular intervals. A case of this type has recently been described by W. L. Birring. The patient was a man 34 years old suffered from an intermittent hydrops with a periodicity of 12 days, the hydropic condition lasting four or five days and coming on without warning and without any apparent cause. Vasomotor disturbances have been mentioned in explanation of this most unusual condition. On the whole, however, these cases are rather mysterious and no satisfactory explanation of this condition has yet been made.

Barlow's Disease.—In Moeller-Barlow's disease the knee joint is frequently involved. This disease is characterized by a subperiosteal hematoma at the lower end of the femur and the upper end of the tibia which causes a periarticular swelling about the knee joint. The joint itself is very painful and contractures of the knee joint are often seen. The diagnosis can be made from the clinical picture, patients being young children which show other symptoms of infantile scurvy, especially anemia, gingival hemorrhages and the skeletal changes of other bones. The *x*-ray picture shows characteristic shadows near the epiphyseal line indicative of the growth changes in the zone of ossification.

Bleeder's Knee.—Hemorrhage into the knee joint is not an infrequent symptom in true hemophilia. The effusion of the knee is occasioned by slight traumatism to the joint and the low coagulability of the blood allows the accumulation of a considerable amount of fluid in the joint. The hemorrhages may lead later to disintegration of the joint surfaces with production of ankylosis. A case observed by the author concerned a young man about 20. He was a true hemophiliac; the knee had ultimately become entirely ankylosed allowing only of a few degrees of motion. The joint surfaces appeared quite irregular in the *x*-ray picture which was due to the disintegration of the articular cartilage.

CHAPTER CXIX

THE CARE AND TREATMENT OF HEALTHY AND PAINFUL FEET

By H. Winnett Orr, M.D., and James E. M. Thomson, M.D.

Lincoln, Neb.

Introduction.—There are very few people nowadays who have normal feet. A few get along fairly well and without much foot discomfort. A great many people, however, have abnormal foot conditions which have become sufficiently serious so that they are not able to do the kinds and amount of work on their feet that healthy and normal persons should be able to do.

One of the things that we learned during our examination of young men for the United States Army during 1917 and 1918 was that about one-fifth of all the young men examined had crippled, painful, or partially disabled feet. All of these had to be rejected or accepted for a grade of service below Class *A*. Even of the men accepted for military service a great many "broke down" in training in this country or upon being required to march, carrying equipment, in France.

By observing the form and use of the feet in small children, and in people who have used their feet without interference by shoes (those who have always lived under natural conditions), we can easily discover that the foot of the average individual in this country has been considerably damaged by modern shoes, hard floors and sidewalks, indoor living, and inadequate care. Most of us have feet which are not even the shape that our feet were originally intended to be. Some of the changes are so marked that we call them shoe deformities. This is particularly true of the toes and sometimes of the arches of the foot.

Failure to take care of our feet properly in the matter of frequent bathing, wearing clean, dry stockings and properly fitting shoes, keeping the feet dry and comfortable in cold and wet weather, etc., is responsible for many of the ailments of which people complain.

It is the object of this discussion to give some instructions in regard to the proper care of the feet under the conditions in which we live; also to give certain general suggestions as to the relief of painful conditions, and the correction and cure of disabilities and deformities of the feet.

Relief and cure of painful foot conditions is much more often possible than is generally supposed. To undertake such cure or relief, however, simply by going back to "sensible shoes" is seldom sufficient. To try that is to "lock the garage after the car has been stolen." Corrective methods must first be used and then proper shoes worn to prevent a return of the trouble. Better than any other way, however, is to prevent such troubles by developing strong feet and wearing correct shoes from the beginning.

In examining a great many people who have painful and crippled feet, we have been astonished to find that so few realize that it is possible to forestall more serious trouble to get relief by correction of these conditions. If one has failed to prevent the development of painful or crippled feet, then it remains to get such relief as may be possible or to cure the condition if that may be done.

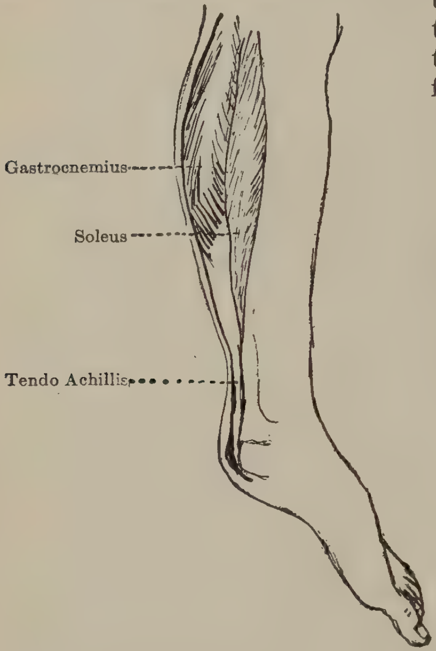

Fig. 249.—Gastrocnemius and soleus pulling foot down (equinus).

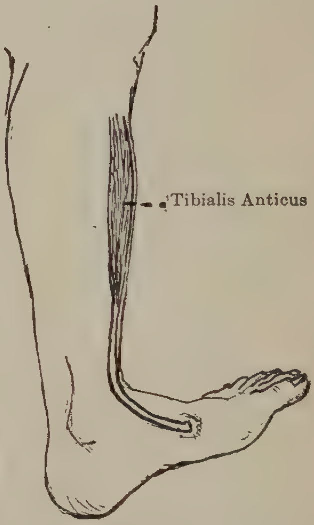

Fig. 250.—Tibialis Anticus pulling foot up (calcaneus).

What people must realize is that a real knowledge of the anatomy and physiology of the foot is essential in the surgeon who is going to treat lame or sick feet. He must be able to make a real diagnosis in foot troubles before he can prescribe treatment that will be effective.

In considering the care of the foot it will be important to bear in mind that in its normal state the bones, ligaments, muscles, etc., have a proper and constant relationship to each other. With any change in that relationship, no matter what the cause, and no matter whether the change be made suddenly, as by an injury, or gradually, as by the wearing of improper shoes, there will be a greater or less disturbance in the proper working together (that is, the function) of those parts and, subsequently, of the foot as a whole. Moreover, foot diseases and disabilities lead to other physical troubles as well. These disturbances in function may and do become in many cases a very

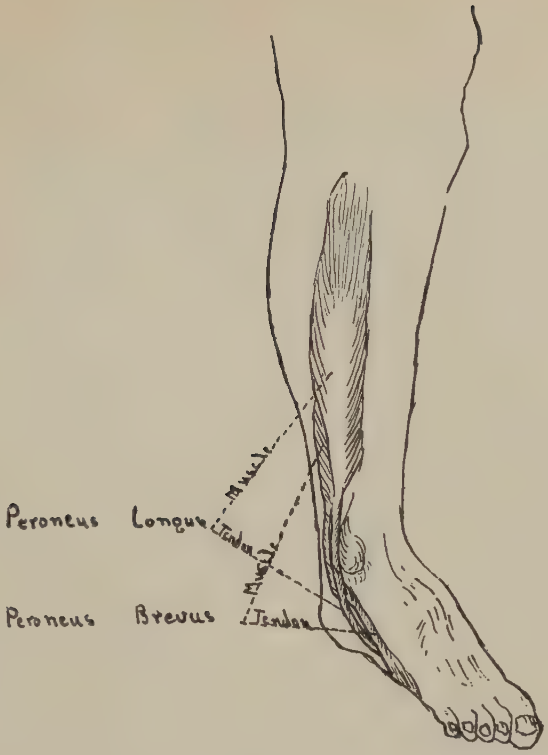

Fig. 251.—Peroneus muscles pulling foot out (valgus).

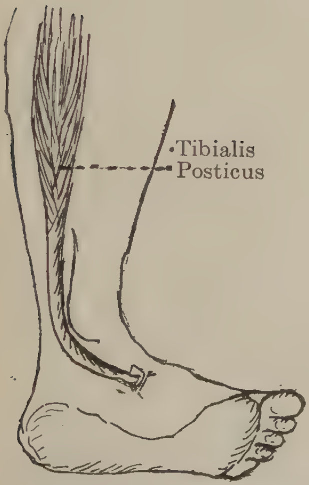

Fig. 252.—Tibialis posticus pulling foot in (varus).

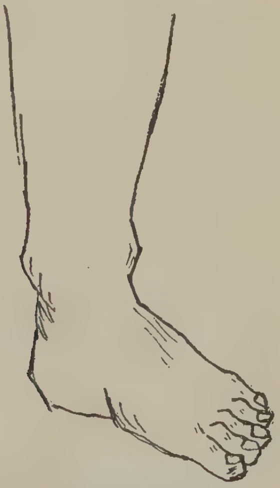

Fig. 253.—Ankle joint too wide or too weak, foot pointing outward (pronation).

serious matter. They will be discussed from several standpoints in succeeding pages.

The working parts of the foot and leg—upon which foot-function depends—may be said to consist principally of: First, the leg muscles that control the foot. Second, the ankle joint. Third, the heel. Fourth, the longitudinal arch of the foot. Fifth, the transverse or forward arch of the foot. Sixth, the toes. At any rate these are the parts upon which the smooth working of the foot depends, and the parts that, when "out of order," are most likely to be the cause of pain and disability.

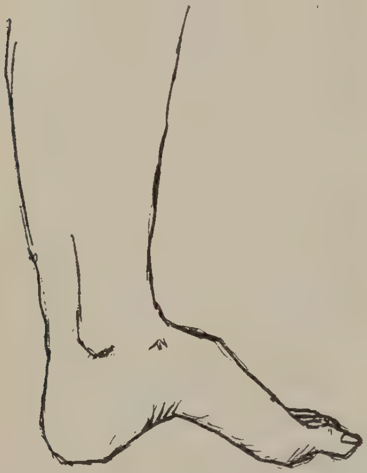

FIG. 254.—Longitudinal arch too short (cavus).

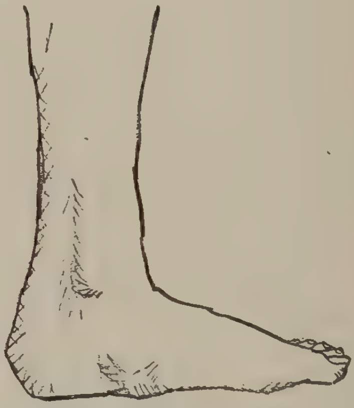

FIG. 255.—Longitudinal arch too long (flat-foot). This condition may be flexible or rigid, note low position of scaphoid.

It has seemed to the authors that a series of carefully prepared diagrams will illustrate the above points better than many words:

Figure 249. Leg muscles pulling foot down too far (equinus).

Figure 250. Leg muscles pulling foot up too far (calcaneus).

Figure 251. Leg muscles pulling foot out too far (valgus).

Figure 252. Leg muscles pulling foot in too far (varus).

Figure 253. Ankle joint too wide or too weak (pronation).

Figure 254. Longitudinal arch too short (cavus).

Figure 255. Longitudinal arch too long (flat-foot). This condition may be flexible or rigid (stiff joints or ankylosis).

Figure 256. The transverse arch may be flat (Morton's toe or metatarsalgia).

Figures 257, 258. Toe deformities:

(*a*) Hammer toe.

(*b*) Bunion (*hallux valgus* or *rigidus*).

Figure 259. Ingrowing toe-nail.

There may be conbinations of these positions such as outward and downward foot (equino valgus) or upward and inward (calcaneo varus).

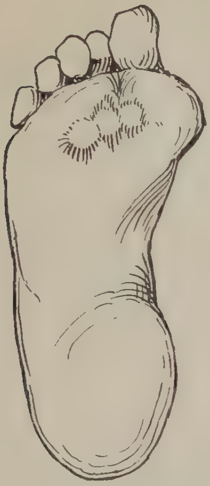

Fig. 256.—Flattened transverse arch (Morton's toe or metarsalgia), calluses under the prominent heads of the metatarsals, contraction of the toes and bunion.

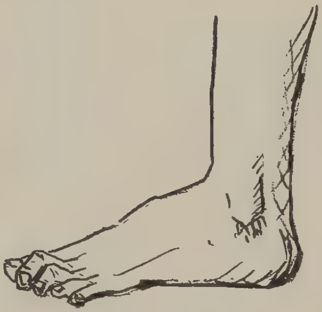

Fig. 257.—Hammer toes.

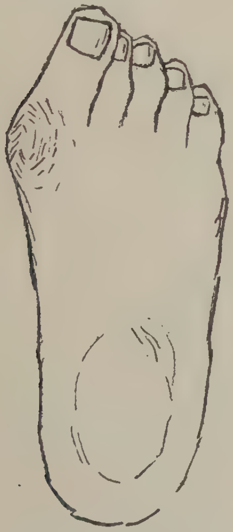

Fig. 258.—Bunion (hallux valgus or rigidus.)

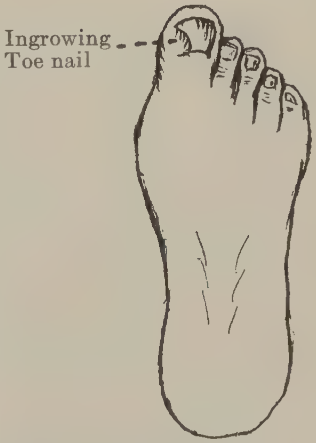

Fig. 259.—Ingrowing toe nail.

Any or all of these positions or changes of position may be temporary and do no particular harm. If, however, an unusual position of the foot becomes fixed or permanent, we have a deformity or disturbance of function to deal with—and pain, lameness, and disability are the result.

It is with these causes and the results of the changes brought about that we shall now undertake to deal.

A few people are born with the feet crippled or deformed (congenital defects). One such condition is club foot, of which there are several varieties. Also we have flat-foot, supernumerary, webbed or deformed toes, etc.

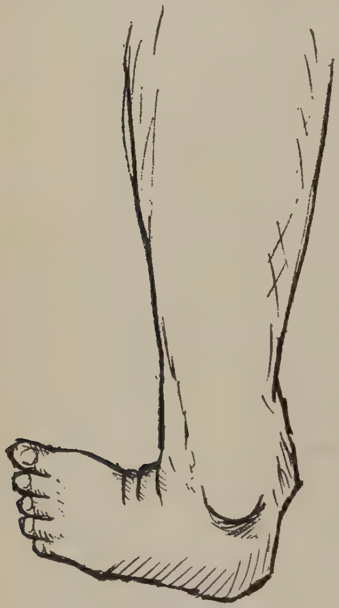

FIG. 260.—Club foot.

Club foot (Fig. 260) is a condition in which the foot is twisted at birth, upwards or downwards, or to one side or the other. The deformity should always be corrected in early infancy if possible. If a child is allowed to begin walking and to continue walking on a club foot, the deformity becomes more and more fixed. Club foot may be cured even fairly late in life, if a suitable operation is performed. It is better for the child and easier for the surgeon to treat a club foot before the child has walked at all. One of the common beliefs in regard to club foot is that this condition can be cured by wearing braces. Braces will hardly ever serve to correct club foot or any other fixed deformity. Braces are usually useful only to maintain correct position of a foot or a leg after that position has been obtained by a surgical operation, or in some other way.

Surgical treatment may be looked upon as practically always necessary to cure club foot. Whether this shall be a manipulation or "bloodless operation," or whether the cords and tendons must be lengthened by an open operation can be decided only by a competent surgeon. It is of the greatest importance that, whichever operation is done, it shall be done by some one specially skilled and experienced in such work. It is quite possible to "spoil" such a foot by unskillful treatment so that afterwards it is impossible to get a good foot by any means.

Flat-foot.—Another condition which is sometimes a birth or congenital condition is that of congenital flat-foot (talipes valgus). When children are born with such flat-feet, or with flat-foot tendency the condition is not usually apparent until they begin to walk. If when weight bearing is begun the feet tend to be flat and the inner ankle rotates toward the floor at the ankle joint (pronation), then it is a very good plan to guard against deformity and disability later on. Such conditions call for support of the foot at a right angle with the leg, and with the foot well inverted by means of adhesive strapping with preceding forcible correction; or with necessary surgical correction with lengthening of the peroneal tendons if the condition is severe. Sometimes also especially in older patients resection of a portion of the tarsus or stiffening of the joints involved in the longitudinal arch of the foot along the lines suggested by Dr. Royal Whitman, Ober, and Steindler, or others, may be necessary. Special shoes and leg braces are of great importance. All of these methods will be referred to in greater detail in the section on "treatment." This condition, if it is allowed to continue untreated, will cause chronic flat-foot; the feet will become stiff and painful, and it will be very difficult for the person to walk about. As a rule, care in the matter of shoes, and heels, and proper exercises will be sufficient in these cases.

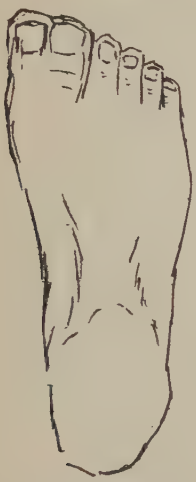

FIG. 261.—Supernumerary webbed great toe.

FIG. 262.—Normal foot.

There are certain other birth conditions and birth deformities of the feet, some of which require surgical treatment and care for their relief. In all these conditions also, the selection of shoes and braces is a surgical problem and should be done under the guidance of, and in consultation with, an orthopedic surgeon who is familiar with such conditions.

Occasionally babies are born with extra toes (supernumerary toes) (Fig. 261). These should be treated early by surgical operation in such a way as to make the feet as nearly normal in shape as possible. In a recent case treated by the writers, the outer half of double great toes was removed in a young woman age 17 and ordinary shoes are now worn with entire comfort.

Webbing of the toes—part or all of them—also occurs occasionally. If the webbing is not extensive it may be let alone. If the deformity is extensive, however, it should be corrected by a careful plastic surgical operation.

Marked congenital contractures of the toes (as, for example, when the second toe is pulled entirely under or the fifth toe entirely over the other toes) are best treated surgically. That is, by the correction removal of the deformed part of the toe. Occasionally, however, persistent stretching or splinting will suffice to correct such a condition.

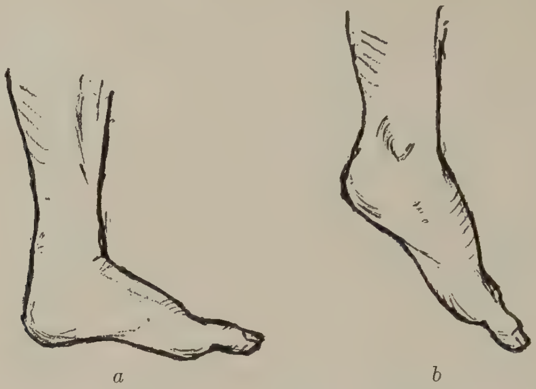

Fig. 263.—Normal foot movements—(*a*) upward, (*b*) downward.

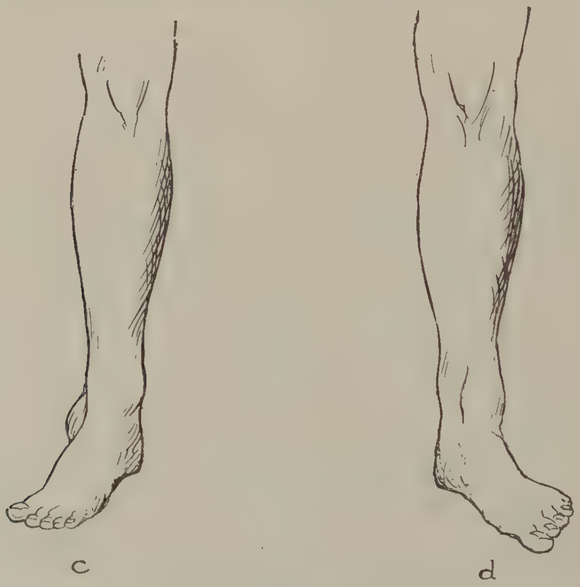

Fig. 264.—Normal foot movements—(*c*) inward, (*d*) outward.

In those individuals who begin life with normal healthy feet, we expect a foot of about the shape shown in the accompanying diagram (Fig. 262). Such a foot may be moved freely and without pain in all normal directions, upwards, downwards (Fig. 263), inwards and outwards (Fig. 264). There is a range of motion that we recognize as normal; the foot being easily movable about so far in each direction. When a patient stands up on such a pair of feet, the position should be more or less as indicated in Fig. 265. The weight is borne on the heel, the outer border of the foot, and the base of the toes. The foot-print of such a foot is as indicated in Fig. 266. If one takes a look at these three diagrams one can see what the shape and appearance of the normal foot should be.

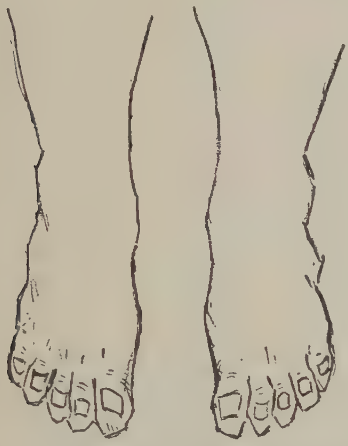

Fig. 265.—Standing position of normal feet, correct position.

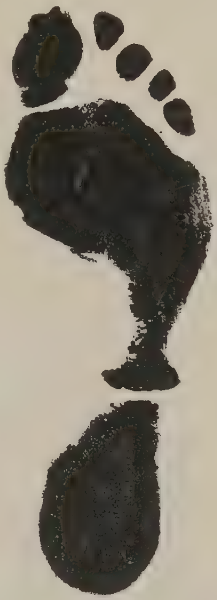

Fig. 266.—Normal foot print.

Figures 267, 268 is that of the average shoe such as most people wear. That this is not a proper shoe for the average foot is apparent at once. A very large share of our foot troubles now-a-days are caused by wearing shoes that are too narrow or too short or both.

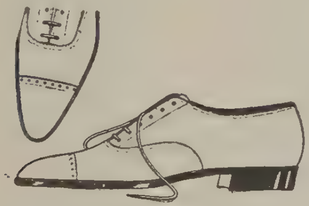

Fig. 267.—Common man's shoe.

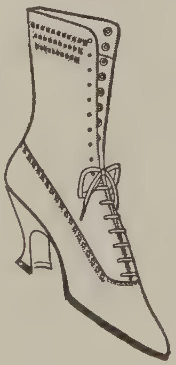

Fig. 268.—Common woman's shoe.

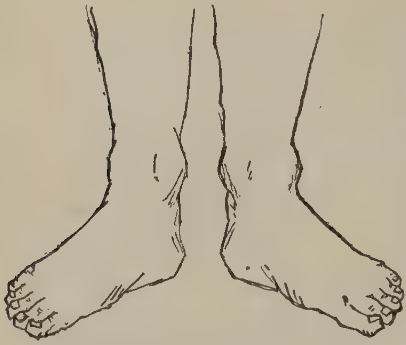

Fig. 269.—Pronated standing position (incorrect position).

There is a popular impression that flat-foot is the prevailing and almost the only important foot disability. As a matter of fact, true flat-foot, as a disabling affection, is rather rare. The painful foot which is commonly called flat-foot is usually "foot strain," instead. This has been emphasized by Doctor Lovett of Boston and others. "Weak foot" which is the name used by Doctor Whitman of New York City is not so good a term because strong feet may be strained as well as weak ones. On the whole whether foot arches are high or low or strong or weak, foot or leg strain may occur and the disability may be very great.

Of course, there are certain other factors that help to cause foot troubles. A great many people suffer from foot strain because of the fact that they are required under modern working conditions to stand for long hours on hard and sometimes uneven floors. The result is that, as the muscles of the feet and legs become tired, the foot settles down into a relaxed position; from being a temporary tired position, it becomes a habitual weak-foot position. Eventually, the muscles, ligaments, and even the bones of the foot become accustomed to this weak or pronated position, as indicated in Fig. 269, and painful, strained, weak foot or flat-foot is the result.

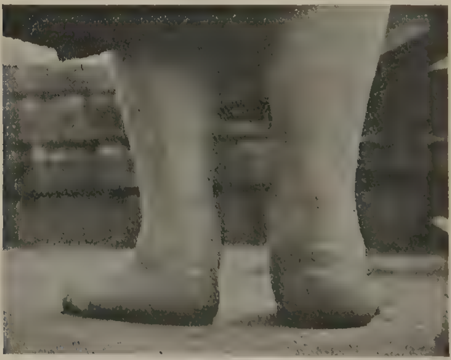

Fig. 270.—Rigid flat-foot showing the rolling outward of the feet on the ankles.

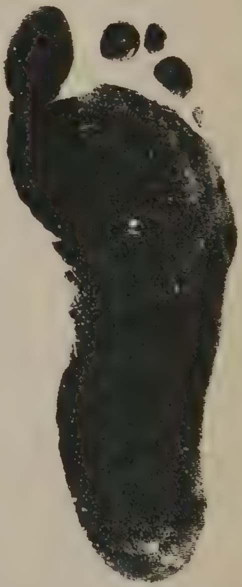

Fig. 271.—Foot print of patient having advanced foot strain.

We sometimes get a similar condition as the result of a rapid increase of weight in any individual. In a person who has gained weight rapidly in a short time, a short period of standing may be sufficient to cause this weak-foot position.

Pronation and chronic strain may be the result. In the same way a person who has been confined to bed for some time by illness or following a surgical operation may upon getting out of bed on the feet, rapidly develop this same condition, and may have weak and painful feet, which cause a great deal of trouble. In treating such feet, relief may be easily obtained if the treatment is begun soon after the feet first become painful and tender. Later on, after the feet become flat and stiff, especially if the disability is very great, relief or cure may be much more difficult to obtain.

Rigid flat-foot is the true flat-foot which occurs after a prolonged period of foot strain, and especially if later there is inflammation (or "rheumatism") of the joints of the foot and ankle. This condition is a very painful and disabling one, and one which calls for the most careful treatment if the patient is to be improved or cured. The picture of true flat-foot is a very striking one and is well illustrated by the accompanying photograph (Fig. 270). The print of such a foot on the floor is the one that is usually employed to illustrate foot strain and is shown by Fig. 271. An important point to remember, however, and one that has already been mentioned, is that this is only one of the causes of painful feet and it is by no means the most common. It is unusual to find feet that are as severely deformed as this illustration indicates. Painful feet, where there is simply foot strain, without much inflammation of the joints is, on the other hand, a very common condition.

In such foot conditions as we have described above, we usually find that there is very poor circulation, and the feet are likely to be cold and clammy and to perspire freely even in cold weather. For this as well as for other reasons, especially in those who are working every day for some hours in a standing position, one should take pains to stimulate the circulation of the feet in various ways. Morning and evening bathing of the feet for a few minutes in hot and cold water, keeping the feet well powdered,* on account of the perspiration, and wearing dry and warm stockings and shoes (which should be changed in the middle of the day if necessary), will all contribute towards preventing such conditions as we have described, or will relieve them after they have once begun to develop.

Weak foot and flat-foot are also commonly caused as a result of a bad standing position, which is assumed by people who have corns or crooked toes, which they are trying to protect by shifting the weight from the outer to the inner border of the foot. This is quite commonly

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* A good powder is

Salicylic acid	2 parts
Boracic acid	8 parts
Talcum powder	90 parts

done, especially if narrow shoes are being worn, and if there is a tender corn on the fifth or little toe. For this reason also, correct shoes which will be described later, ought always to be worn, especially by those who must depend upon comfortable, strong feet in their every day work (Fig. 272).

So far one may say that there is practically only one suitable last for the manufacture of shoes to be worn by people who have painful or disabled feet. This is the so-called army last, which is shown in Fig. 272. This last may now be obtained in a number of shoes made by the best factories. There are still being sold, however, thousands of shoes which are too narrow and which are constructed along improper lines otherwise. Such shoes are responsible for many corns, bunions, ingrowing toe nails and other foot troubles. A great deal of foot disability is produced or encouraged also by the improper fitting of shoes (Figs. 273, 274). This subject will be referred to again, but one may say that the correct shoe is one in which neither the toes nor the broad part of the front of the foot are crowded or pushed to the outer side. The front part of the shoe should be straight along the side of the great toe, and the entire front of the shoe should swing slightly inward. In any foot also in which there is a tendency for the weight to be thrown on the inner border of the foot, this should be overcome by the way in which the shoe is built, or by a special heel which will tend to throw the weight of the foot onto the outer border of the heel and the foot.

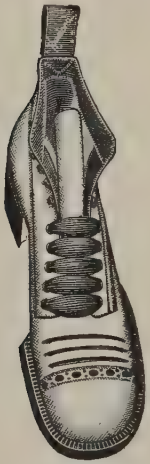

Fig. 272.—Munson last shoe.

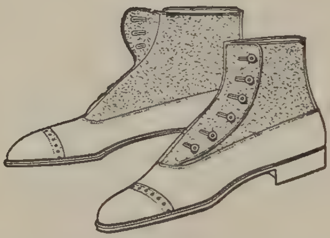

Fig. 273.—Incorrect men's shoes.

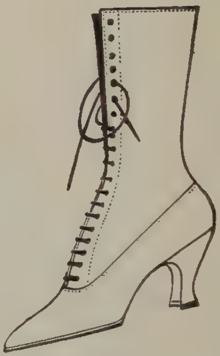

Fig. 274.—Incorrect woman's shoe.

One is frequently asked about the correct standing and walking position of the foot. The answer must always be that in standing the feet should point very slightly, if any, outward (Fig. 265), and in walking the feet should point pretty well to the front (Fig. 275) or directly forward. The rather common custom of pointing the feet outward at an angle of 40 to 60° as used to be done in the "correct" military or gymnastic position of "attention" (Fig. 269) is quite wrong (Fig. 276). Of course many of our modern shoes are so built that this position is necessary to preserve one's equilibrium, but both the position and the shoes should be changed for the sake of the feet.

One of the next important subjects to consider is that of deformities of the toes. The congenital (birth) deformities of the toes have already been referred to. Contracted toes, webbed toes, over-lapping toes, etc., in infants can occasionally be corrected by a prolonged course of holding them in correct (or over corrected) position with adhesive tape or by splints. As a rule, however, surgical treatment is indicated and, if properly done, will give immediate and satisfactory results. There are several other varieties which are usually caused by injuries or by shoes that are too narrow or too short, or both. The commonest manifestation of such trouble is *corns*. These occur usually on the outer and upper side of the fifth toe, or on the tops of the second, third or fourth toes, or in the form of what are called soft corns between the fourth and fifth, or the third and fourth toes. All of these conditions are caused by pressure. In the so-called soft corns between the toes, the pressure is indirectly caused by bone pressure against the inner side of the toes (Fig. 277) because the shoe is too narrow across that part of the foot.

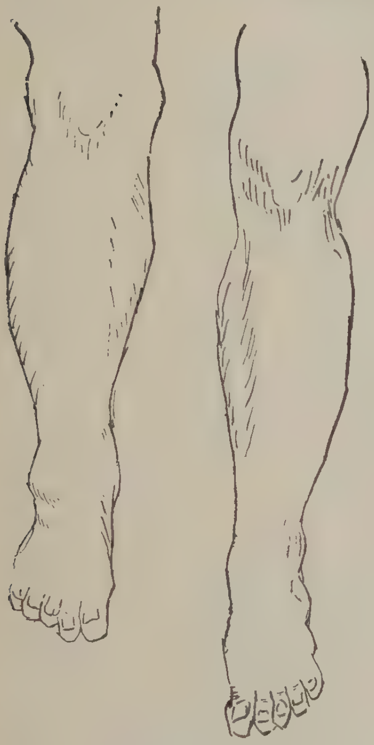

Fig. 275.—Correct walking position.

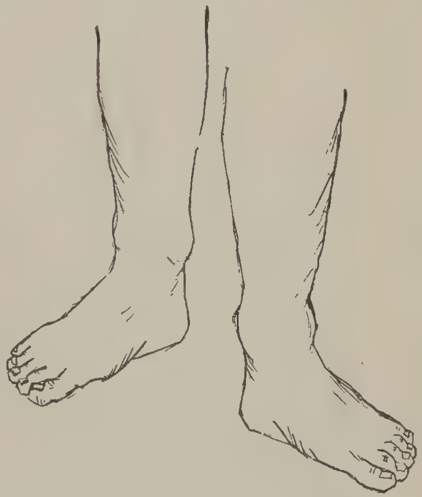

Fig. 276.—Incorrect walking position.

The other corns are caused, of course, by bone pressure underneath the skin against the inner side of the boot or shoe, so that there is not sufficient room for the toes to spread out in the direction they should.

Corns themselves are painful because of the irritation of the nerves that lie in the skin at the point where the corns are produced. But this is only part of the discomfort and disability that arise out of such a condition. As explained in a later paragraph a painful corn affects the entire foot so that there is a tendency on the part of the foot and leg muscles to draw and distort the foot, and there is also a disposition to adopt improper standing positions, and cause foot and leg strain and other disabilities.

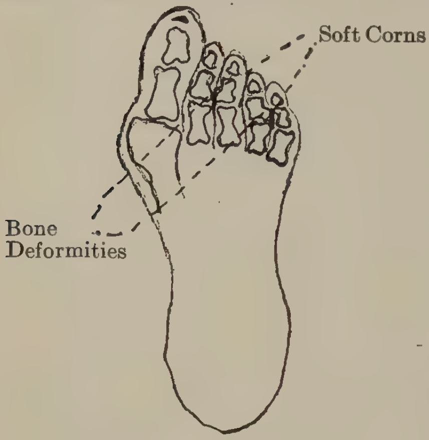

Fig. 277.—Site of soft corns due to pressure of deformed bony prominences.

Corns may always be prevented by wearing correct shoes in the first place. If they develop and become sufficiently severe, however, the cure of such conditions cannot be brought about even by the change to correct shoes. We sometimes get in corns, as in bunion, an actual enlargement of the bones under the corns For that, an operation is necessary to remove the enlarged and irritated portions of the bones. This treatment is comparatively simple now-a-days, because, in the first place, the operation may be done under local anesthetic so that it is a painless procedure, and secondly because we now understand the principles of doing clean surgery, in such a way that, if properly done, there is no danger of inflammation or infection after such an operation. It is important to remember, however, that in the hands of unskilled and inexperienced surgeons these operations may result in inflammation, infection (blood poisoning) or other disagreeable complications.

Corns are always caused by improperly fitting shoes, sometimes because shoes are too narrow, and sometimes because they are too short. An important thing to remember, also, is that any painful condition of the foot may result in a drawing of the entire foot; sometimes the arch draws up, and sometimes the foot draws outward so that the arch is flattened. This condition is spoken of as muscle spasm; it is caused by any painful or irritating condition, and the result is for the irritated and painful muscles to draw up and distort and change the position of the foot. If this keeps up for a very long period, permanent contraction and drawing and deformity of the foot will be the result.

Hammer toes are also caused in the same way. A shoe that is worn too short habitually will cause the toes to draw up and the first joints to become irritated by pushing against the top of the shoe. In a short time the irritated or spasmodic muscles underneath will become tender and draw the toes as indicated in the diagram (Fig. 257). This is the condition also, which if not relieved early by wearing proper shoes, becomes a chronic affair, and an operation similar to the operation for corns is necessary for a cure.

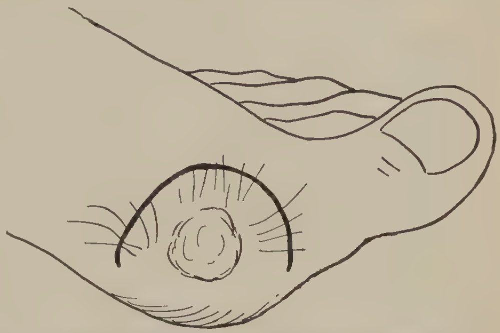

Fig. 278.—Incision for bunion operation.

Ingrowing toe nails (Fig. 259) are likewise the result of short or narrow shoes. It should always be understood that the nail *does not* grow in at all. What happens is that the soft tissue grows *over* the nail. The common operation of removing part of the nail should never be done. Instead, the extra soft tissue including the skin along the edge of the nail should be carefully removed and then correct shoes should be worn. All cases can be cured in this way. Attention should also be given to the matter of trimming the nail straight across instead of in a curved line, as is more commonly done.

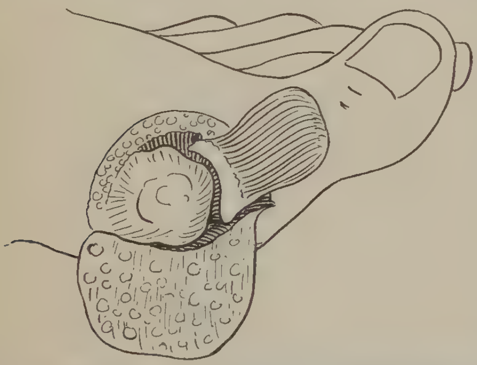

Fig. 279.—Head of bone exposed bursal flap turned back.

Bunions (Hallux valgus) deserve to be treated in a chapter by themselves. They are brought about in the same way as corns and resemble corns in many respects. However, the condition is so common and the deformity and disability so serious that special consideration of the subject must be given.

Bunions, as hammer toes, are caused invariably by wearing a shoe that is too short or too narrow; the first or great toe is crowded over towards the middle of the foot (Fig. 258). This is in conflict with the original design of the foot—the first toe is designed to point more or less straight forward. After a period of irritation and muscle spasm, and especially if the shoe is rubbing against the base of the first toe, we have a toe that is drawn over towards the middle of the foot and drawn downwards, and have a second joint that is enlarged, irritated and tender. In such a case the correction of the foot wear is of the greatest importance. If the condition has advanced so far that relief may not be obtained by the adoption of correct shoes, surgical treatment as described later is necessary.

This bunion deformity may be of any grade of severity. At any time, however, after the metatarso-phalangeal joint of the great toe has become so much enlarged that it is conspicuous even through the shoe, or when the joint is so inflamed so that it is always tender and painful, the condition is a surgical condition and one need not expect relief by treatment in any way except by operation.

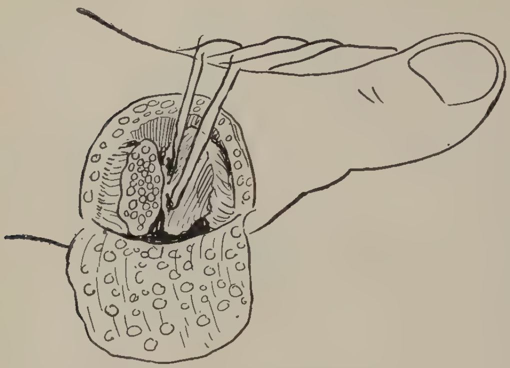
Fig. 280.—Head of metatarsal removed, bursal flap sewed into base of wound.

The treatment which has been used for some years, and which is almost always successful in relieving this trouble, is to remove the enlarged and sensitive portion of bone just behind the second joint of the great toe. This operation is also now commonly done with local anesthetic, just as teeth may be painlessly removed in the same way. It is necessary for the patient to be laid up for four or five

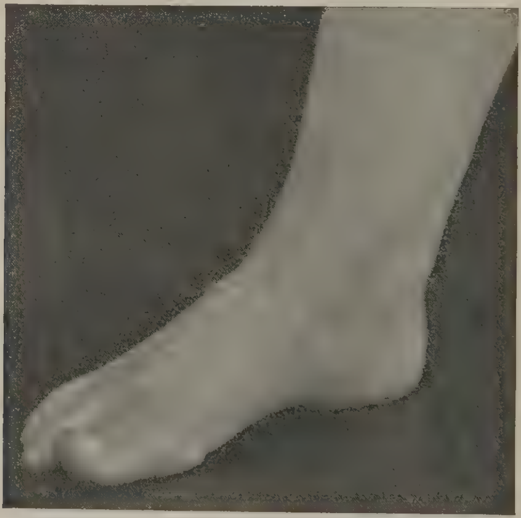
Fig. 281.—Foot 10 years after bunion operation.

days or a week; after that one can get about quite well, and at the end of two weeks, as a rule, the patient may resume his or her regular occupation unless it involves heavy work in the standing position or too much walking. The relief from a suitable operation of this kind is very great. In fact, we have very few more appreciative or grateful patients than those for whom a bunion operation has been done.

The great toe, while it may be slightly shortened, is made straight and movement is more easily possible. The enlarged, painful, bony portion of the bunion is removed. The question is frequently asked as to the danger of making a stiff toe. This practically never occurs except as a complication, which should be avoided. There is a condition somewhat like bunion in which we have a stiff joint (hallux rigidus), but this condition is more likely to occur in patients for whom no surgical treatment has been given.

THE KINDS AND CAUSES OF FOOT TROUBLES

Before consulting a surgeon or physician regarding painful foot conditions, patients have usually exhausted the resources of special shoes, arch supports and chiropodists. The failure of such methods of treatment is for the reason that successful treatment of foot troubles can usually only be given when the foot itself and the causes and character of the foot trouble are thoroughly understood by someone competent to do so.

Painful feet are just as often diseased feet as are painful organs in any other part of the body. Slight, as well as painful, foot affections are to be looked upon as requiring intelligent diagnosis and careful treatment. The factors entering into the causes of foot troubles will be discussed in this chapter from all the standpoints commonly used in considering other physical ailments. That is to say, we shall undertake to lay down here certain principles of diagnosis along the same lines as those by which other diseases are diagnosed and plans made for treatment.

Age.—Painful foot conditions are of course most common in adults. Even in infancy, however, we have certain foot conditions that must be recognized and carefully treated if the individual is to develop strong, useful and comfortable feet and legs.

Congenital club foot and flat-foot have already been referred to. One of the common causes of foot deformity in childhood is rickets. In this disease the foot troubles may manifest themselves simply as weakness of the muscles and ligaments with corresponding foot strain, or there may be actual change in the bones of the feet and legs so that we have severe pronation of the foot with or without bow legs or knock-knee. Many times a mother will bring her child, saying that the child becomes tired easily and cries to be carried after walking for a short distance. This may be true even in children who have no apparent deformity of the feet. If there are changes in the shape and form of the feet and legs along with this tendency to be overtired on slight provocation, special methods of treatment should at once be instituted, which usually afford relief.

Foot strain is also very likely to develop between the ages of 12 and 18, at which time boys and girls begin to be engaged in occupations which involve more standing, or the carrying of heavy loads.

Young mothers are also very prone to develop acute foot strain. After this time, age is not likely to be a factor until from 40 to 50, at which time those individuals who may have been having some foot strain for a good many years are likely to settle down into a chronic flat-foot position. If such an individual has also been carrying a certain amount of focal infection in the mouth, tonsils, gall-bladder, appendix, or elsewhere, arthritis may supervene at 40 to 45, and painful, rigid, flat-foot may be the final result.

Sex.—Sex is not a determining factor in flat-foot except as it has to do with occupation or special conditions to which the individual is subject. In other respects, however, it may be said that corns, bunions, hammer toes, metatarsalgia and such other conditions are much more prevalent among women than among men. This is obviously for the reason that the wearing of shoes too narrow and too short is more common in women. Motherhood tends to foot strain during and after the period of pregnancy. The prescription of prophylactic foot wear for prospective mothers is of the greatest importance, though one may say that it rarely receives attention.

Occupation.—Occupation is a factor of first importance in considering foot conditions. Those engaged in nursing, clerking, as barbers, waiters, policemen, and in other similar occupations are especially prone to foot strain, flat-foot, and the allied conditions. Such foot disability is obviously the result of overuse of the feet. It is not uncommon to find that a patient with a painful foot condition has changed his occupation even before consulting a doctor because he found that he could not continue in work which required much standing or walking.

Habits and Constitution.—Habits are sometimes a factor of considerable importance. One interesting condition which has been called to our attention in recent years is that of "pulseless feet." This occurs principally among members of the Jewish race who are immigrants to this country. It has been found that the condition is likely to be associated with excessive cigarette smoking and tea drinking.

Antecedent History.—It has been noted in numerous instances that grandparents, uncles and aunts, father or mother, and several children , all suffer from feet that not only look alike, but that develop similar degrees of foot strain and other kinds of foot discomfort. This predisposition or this tendency is almost sufficiently pronounced to be rated with the diatheses and must be taken into account in promising relief or cure in certain cases.

Personal History.—The amount and degree of disability in certain individuals must be carefully studied in order to decide upon methods of treatment and the predictions that are made regarding relief or cure. If there is an acute foot strain, a few weeks following an illness, or after subjecting the feet to an unusual amount of work, one may expect prompt, fairly complete relief from simple methods of treatment. If, however, as frequently happens, the individual presents himself with severe disability which has lasted for several years, much more radical treatment will be necessary, prolonged after-care, and a guarded prognosis.

Mental and Moral States.—The patient's attitude toward his trouble and his willingness to coöperate in the treatment are of the greatest importance in deciding about his condition and his prospects. If a foot condition is to be relieved by exercises, by any regimen as to diet, etc., the patient's mental and moral conception of the situation and his coöperation are or may be of the greatest importance. There are certain conditions also which are either minimized or greatly exaggerated by the mental attitude of the patient.

One does not look for hysteria to show itself in the feet, and yet the authors have noted several cases in which painful conditions of the feet proved to be practically or entirely hysterical in origin.

Social Condition.—Whether a patient is constantly occupied or comparatively at leisure, has an important bearing upon one's estimation of foot conditions. The treatment must also, of course, be prescribed according to the amount of time and effort the patient will be able to give in contributing to his own recovery.

Duration of Disease.—If the symptoms of which the patient complains are of recent origin, he may be suffering simply from an acute strain or a temporary condition, which may be easily and quickly relieved. If, on the other hand, the disability has existed over a long period, or if there have been several attacks, questions of strain, infection, arthritis, neuritis, etc., can be decided more easily after information in regard to the above and other points is at hand.

The possibility of new growth (sarcoma or epithelioma) will have to be decided partly upon the duration and history of the trouble, and examination of the patient. The *x*-ray is invaluable in such cases but, contrary to a general notion, not of much help in deciding about foot strain.

Residence.—Where a patient lives or where he has lived and under what circumstances must often be carefully considered in arriving at a decision in regard to his condition and relief. This has to do especially with the character of infections to which he may have been exposed and the prospect as to whether any arthritis or neuritis that exists is of external or internal origin. If there is a question in any case as to a possibility of a tuberculous or syphilitic bone or joint infection in the foot or ankle, that must also be decided partly upon the circumstances under which the patient has lived and the possibility of his having been exposed in his home or place of occupation.

Posture or Attitude.—Many foot conditions may be recognized promptly simply by looking at the patient as he stands without even removing the shoes and stockings. In advanced rigid flat-foot the patient has what is known as "wooden gait." The feet and legs move together as if they were carved out of a solid mass. This is a combination of bones and joints that have become rigid and the foot and leg muscles which are in habitual spasmodic contraction. There are of course many degrees of this condition, but the appearance is quite characteristic even in the early stages. Later on the condition may be recognized even at a distance.

Other conditions about the feet become more apparent only upon inspection of the feet themselves, and the attitude of the patient after the shoes and stockings have been removed. Lesser degrees of flat-foot or pronation are perhaps apparent only or chiefly because of the downward displacement of the head of the astragalus, the flaring outward of the front of the foot (see illustration), and the tendency of the tendo achillis, as seen from behind, to bow out, rather than curve in as it should.

Contracted and **deformed toes** are apparent at once, but neuritis (metatarsalgia) calls for more careful examination as a rule. Certain other conditions, as for example, a strain of the plantar fascia on the sole of the foot, may give a patient who bears weight only on the toes or the heel according to whether the place of tenderness is at the front or back. In certain cases in which patients have large painful callouses under the base of the toes (Fig. 256) or under the heel, there may be a tendency to bear all of the weight on the outer border of the foot. In the flat-footed condition shown, however, there is a tendency for the weight to be borne on the inner border of the foot. In such cases the patient is likely to walk with the feet turned well outwards (Fig. 276). This same attitude, which has been commonly taught in physical education and gymnasium classes, is an attitude of weakness and has in many cases contributed to the strain and breaking down of patients' feet.

Mobility of the Parts.—As has been mentioned before, a free range of motion of the foot in the four principal directions (Figs. 263, 264), upwards (dorsi-flexion), downward (plantar flexion), inward (adduction) and outward (abduction), in general may be said to indicate a normal and strong foot with good leg muscles. Even such a foot, however, may be easily strained by prolonged standing or by walking considerable distances on hard side walks or with heavy loads.

As soon as symptoms of irritation or inflammation occur, however, there is a change in the range of motion of the foot. With inflammation we are likely to have restricted movement in one or all directions of the foot, and movement becomes painful and uncomfortable. This is due to what we call muscle spasm. The condition is brought about by the unconscious reflex contraction of the muscles which automatically and even unconsciously limit the movement of painful or sensitive parts about the affected joints. In acute flat-foot or foot strain for example we frequently have a considerable spasm of all the foot and leg muscles. Sometimes the spasm of the peroneal (abductor) muscles is so pronounced that the tight cords are noticed above and behind the outer ankle (external malleolus), also in such cases sometimes at night there is pronounced cramping (muscle spasm), and these contractions, even at night after the patient has gone to bed, may be so severe as to keep him awake or awaken him after he has gone to sleep.

Muscle spasm frequently manifests itself by aching and pain in the thighs, hips, or back. One of the striking effects of muscle spasm also is the stiffness or lameness in such patients when they first try to walk after sitting for a time or on first rising in the morning. The stiffness is sometimes so severe that quite a time must be spent in "limbering up" before such a patient can get around well. This is the soreness and stiffness naturally resulting from a day of strain or "muscle spasm" the day before. Later on, if there is developing a condition of chronic strain, there may be in addition to this transitory stiffness a semi-permanent muscle spasm and joint stiffness also, so that there is a great deal of fixation about the entire foot, ankle and leg, and the patient walks with a very stiff, unnatural, wooden gait.

In paralyzed conditions about the feet we may find paralysis of any single muscle or muscle group. Loss of power in the muscles in the front of the leg, for example (anterior-tibial), results in a drop foot condition. Under such circumstances the muscles at the back of the leg become shorter, and while the muscles at the front are unduly relaxed, those at the back are tighter, and a contracted foot with the toes downward (talipes equinus) is the result. Similar deformity may take place in any direction, with the paralyzed muscles letting the foot go, and the strong muscles shortening up and pulling the foot in that direction. Very advanced conditions of this sort can only be corrected by surgical operation as will be described later.

Temperature of the Feet.—This is an important point to be considered in dealing with any conditions about the feet. They are especially subject to variations in temperature on account of the fact that the blood supply is so far away from the heart and the large central vessels. In any acute condition about the feet, especially if there is inflammation, the feet are likely to contain more blood (congestion) than usual. Such feet are hot to the touch, and feel hot to the patient. Even in acute foot strain without true inflammation, this is quite likely to be the case. One frequently sees patients with recently developed acute, painful feet in which the congestion is very apparent. The feet appear swollen and red, the veins which are immediately under the skin are large and blue and engorged with blood returning toward the heart.

If there is acute trouble in the joints (such as is commonly called "rheumatism") the feet may also be swollen, tender and hot. After a time, however, if the condition settles down into one of chronic foot strain or flat-foot, the feet are cold and the circulation poor. In such feet there may be a tendency toward excessive perspiration and a sort of clamminess that adds to the patient's discomfort and disability. In certain of these conditions the feet may be extremely pale or of a light-bluish color indicating the impaired and enfeebled character of the blood supply. In certain instances there may be spots of discoloration on account of ecchymosis.

Size and Shape of the Feet.—This is a matter of considerable importance in examining the feet. The disabilities of the feet depend to a considerable extent upon their size and shape. Feet are by no means strong in proportion to their size. Some large robust people with very substantial looking feet nevertheless have extremely painful and uncomfortable members. The long narrow foot is very likely to be a weak foot. Short thick feet, if they become strained or irritated, are more likely to develop the contracted deformity like hollow foot, hammer toes, etc.

In general one may say that examination of the feet frequently gives an early clue to constitutional diseases or even local diseases situated in other parts of the body. This is true, for example, with reference to patients who present themselves with an arthritis of the feet which may have had its origin in infected teeth, or tonsils, or in some other focus. Quite commonly too much emphasis is placed upon this point. The authors have seen many patients for whom teeth and tonsils have been removed with great enthusiasm with a design to cure a well established arthritis. Such conditions are often local and can be relieved by careful treatment of the feet alone. There is more to be said in regard to this point.

Circulatory Disturbances.—Circulatory disturbances which have their principal seat in the heart or the great vessels frequently go unrecognized until foot swelling (edema) or other foot and leg symptoms lead one to investigate the patient's general condition. The same may be said with regard to kidney and other conditions. Lead poisoning is sometimes first seen on account of foot- or wrist-drop. Diabetes may be brought to the surgeon's attention through a gangrenous toe, and there are other infections of the skin, nails, etc., of the feet that lead to a diagnosis of chest or abdominal conditions. Forms of spinal cord disease with impending paralysis or other nervous disturbances may give their earliest manifestations in the position, strength, movements or usefulness of the feet.

It is of the greatest importance to interpret rather carefully the patient's history of the character and extent of pain. The writer of these lines has seen hundreds of patients who have consulted physicians on account of painful feet, and in spite of the fact that careful accounts are given of foot and leg symptoms and disabilities, the physician only occasionally makes the sort of investigation he would make if the pain were located in the abdomen or chest. Foot pain and foot disability are often as distressing to the patient and as disabling, as far as occupation or recreation are concerned, as pain anywhere else. And the patient is equally concerned about treatment and relief.

Early and Adequate Recognition of the Character of Foot Troubles.—Taking into consideration the points mentioned above, foot disability is a matter of sufficient importance to so many patients that much more attention should be given to these problems than usually is. To dismiss this class of patients with a wave of the hand to the uninformed shoe salesman, or to the badly informed chiropodist, is scant justice considering how many such apply to the members of our profession in the first instance for relief.

From the preceeding it will be apparent that the best kinds of treatment for the feet are those which are applied early and which provide against the development of the more severe conditions which have been described. This is true for flat-foot, bunions, hammer toes, and all the rest. As we discuss the treatment of these conditions, it will be pointed out how early preventive measures could have been used so that other more serious therapeutic measures or methods of treatment, especially surgical, might have been rendered unnecessary.

Prophylaxis, Mechanical and Surgical Treatment.—It is a mistake to suppose that because these conditions have not been prevented, or have become severe, treatment will not give relief. We have seen some of the most distressing and painful foot conditions possible entirely relieved by careful treatment, or by surgical operation.

Club foot (Fig. 260) and the other congenital defects of the feet commonly require surgical treatment or surgical operation to cure. If the condition is very mild, however, and a very thorough series of manipulations is carried out beginning when the baby is a few days old, club foot may be sometimes entirely cured and a normal foot secured by the time the baby is old enough to walk. The decision as to the kind of treatment to be employed must always be made by a competent surgeon. Moreover, when surgical treatment is recommended it should always be carried out by someone with previous experience in the treating of that particular condition. Many feet are damaged by the attempts of amateurs to treat them surgically, by plaster casts, by braces, or otherwise. Our previous statement with regard to braces applies particularly in the case of club foot, namely, that braces cannot be used to correct deformity. They are only useful to maintain correct position after it has been obtained by surgical operation or in some other way.

Another point to be borne in mind is that club feet should be fully corrected in time for the baby to begin to walk with his feet flat on the floor. The difficulty of correcting club feet becomes greater as soon as the child has been allowed to walk on the feet. Even then, however, if the baby's feet have been neglected, the parents should not hesitate to undertake treatment of an old crippled foot. With our modern methods, even a foot that has been walked on for several years can be practically entirely cured. It is better, however, for the feet to be treated early and before the time for walking comes on.

One of our great modern teachers in orthopedic surgery, William Little of London, had his club feet operated on after he was practicing surgery. His feet were so greatly improved that he became one of our most enthusiastic and most successful orthopedic surgeons.

There has been a good deal of discussion as to the exact details of the method to be used in operating upon a club foot. In general it may be said that it is important to fully over-correct the foot either by manipulation, or by open operation with as little damage to the bones and joints of the foot as possible. Careful lengthening of the contracted tendons and possibly the removal of the astragalus are usually sufficient to permit full correction. In the very rare instances in which a more radical operation is necessary, it will have to be left to the discretion and judgment of a competent orthopedic surgeon, both to decide upon and to perform the operation. This remark applies particularly if there is a crippled foot in which the attempt has been made one or more times to secure correction of the deformity without success.

Weak foot, foot strain, pronation of the *foot*, and *flat-foot*, may all be considered together. We have here several conditions which in some cases combine to cause a great deal of distress and disability for the patient. In children we frequently have flat-foot without very much strain (Fig. 282). The foot may be badly pronated (so that the weight is thrown on the inner border of the foot) and the child may walk badly and get tired easily without very much discomfort otherwise. In older patients, on the other hand, we frequently have men or women with very painful, badly disabled feet, without any appearance of pronation or flat-foot whatever. This is for the reason that in the latter case we have a true foot strain with slight displacement of the bones of the foot, a good deal of ligament strain, and muscular spasm of the muscles of the leg, when the arch of the foot has only begun to give way, and when there is as yet no deformity that can be seen, or very little.

Later on either in children or in adults the arches of the foot (longitudinal or transverse) may break down entirely, there may be infection or inflammation of the joints, and finally, stiffening (ankylosis) so that there is a true, rigid, flat-foot, which would never have developed if the earlier symptoms had been recognized and treated along the lines which we shall suggest. It is in the early stage of this condition that full correction of the feet to a right angle with the leg and inversion of the foot for a time gives so much relief. The feet should be kept in plaster of paris in this position for at least three to six weeks and the patient should walk in the casts. This is often *not done* for fear of stiffness but it is a fact that much stiffness can be avoided in just this way.

If, as we may suppose, the first symptoms of foot strain are due to weak foot, and the result of a tendency on the part of the arches to break down, the indications for relief are fairly clear. That is, some artificial support must be given to assist the muscles and ligaments that are unequal to the burdens that are being put upon them. Because this is so obvious to everyone, arch supports, foot plates, leather bands, shoes with built in supports, and all kinds of patented or mechanical contrivances that look as though they would support the foot have been sold at fancy prices and at great profit to the manufacturers. And yet these appliances are only occasionally really beneficial. Patients who come for treatment are very frequently those who have had considerable experience with special shoes, arch supports, etc., and who have not yet obtained any particular relief. This is for the reasons that have been spoken of before, that the degree of trouble as well as the shape and size of the foot are subject to the greatest variations. No two feet, not even those of the same individual, are quite alike. A method of treatment, therefore, which cannot be readily adapted to the foot under consideration is usually inadequate. Moreover, every method of treatment used in such cases should have in mind the real restoration of the foot, that is, we should try in every case to convert a weak foot into a strong foot if possible, so that mechanical appliances will not be necessary. This is nearly always the object we now have in mind in treating feet of this sort.

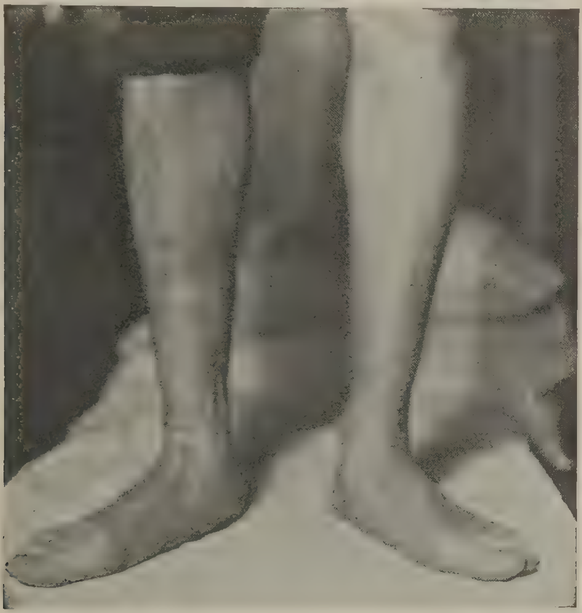

FIG. 282.—Flat feet, congenital in adult 40 years of age; father, grandfather, and brothers have like condition of feet.

Our methods of treatment, therefore, are somewhat as follows: First of all in the mild acute cases, temporary support for the foot by means of a special method of adhesive strapping (Figs. 283, 284) followed by a course of exercises for the foot and leg, will usually serve to "cure" that particular case.

For the patient who shows a disposition to more prolonged or more chronic trouble, we usually adopt at once the so-called Thomas heel. This heel is of a special shape and size and is built entirely upon the outside of the shoe (Fig. 286). Controlling the position of the heel is very important in all cases of weak or flat-foot, because the heel has been found to be the weak point in most of these cases. Even very early in the development of flat-foot it will be observed (Fig. 287) that the heel tends to flare outwards, rather than inwards as it normally should. The effect of the Thomas heel is shown in the illustration (Fig. 288) to correct that deformity, and to correct it in such a way that in walking every step tends to strengthen rather than weaken the muscles on the inner side of the foot and leg.

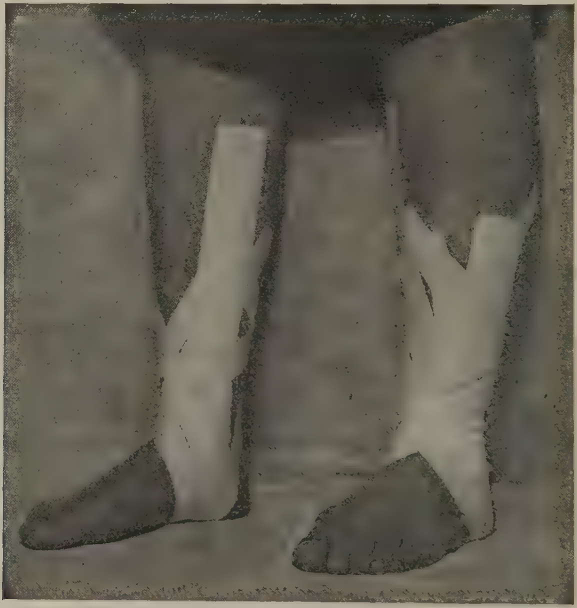

Fig. 283.—Basket weave strapping. Side view.

An interesting experience in the prevention of foot disability was that of Dr. R. B. Osgood* whose conclusions follow: "In conclusion we wish to advocate the preventive treatment of foot strain among classes of persons beginning to assume occupations calling for a greater number of hours on the feet under conditions which preclude intermittent rest. Policemen, firemen, ward tenders, and trained nurses are among these classes. Our experience has been limited to the nurses at the Massachusetts General Hospital and has covered a period of over two years. The number of probationers examined has been 133. Before these preventive measures were adopted it was at least a very common thing for us to be called upon to treat severe cases of foot-strain, and the number of nurses laid off in the course of a year on account of foot symptoms was an economic waste appreciably felt. All probationers have for the last two years been examined during their first few weeks of hospital life by the

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* Journ. Orth. Surg. ix (1911), p. 416.

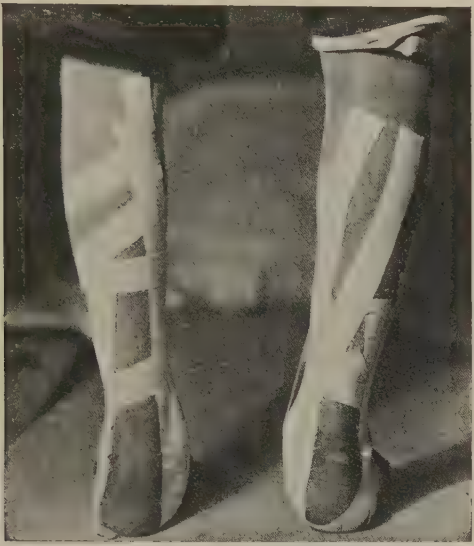

FIG. 284.—Basket weave strapping, view from back.

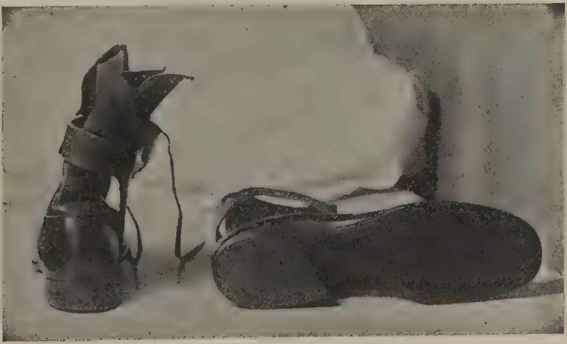

FIG. 285.—Thomas heel.

methods described in the earlier portions of this paper. They have had explained to them the reasons for wearing during their working hours proper shoes. Lasts suitable to their individual needs with and without Thomas heels have been prescribed. Eighty-four of these 133 were considered to need advice. The simple cases of strain and those who were believed to possess the potential of strain have been given this advice at the time of first being seen. The more severe cases have been told to report to the Out-patient Department and appropriate treatment has been more carefully planned. Sixty-four showed an abnormal muscle balance. Nineteen reported to the Out-patient at some period of their course complaining of foot symptoms. Twelve, or 60 per cent., of this 19 reported by request after the first examination, were treated, and never more heard from. Seven reported for trouble later in their course. Of these, two were cases of infectious arthritis, one was acute strain of the anterior arch,

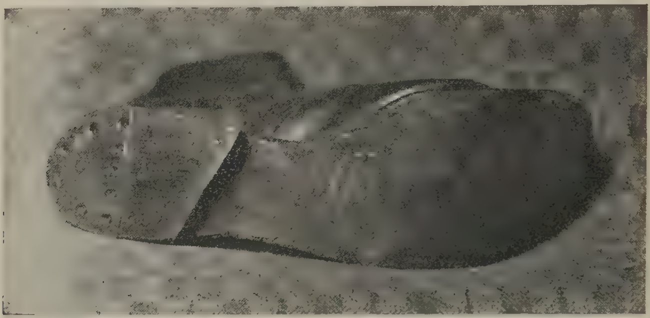

Fig. 286.—Thomas heel with hard rubber used on the back part of the face. This substance wears much longer than leather and allows the patient to walk more quietly.

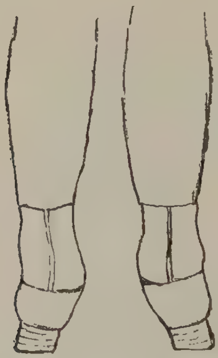

Fig. 287.—Characteristic attitude in pronation or flat feet.

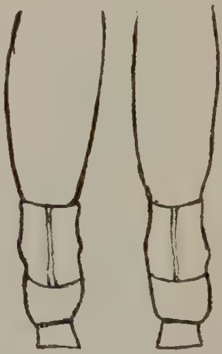

Fig. 288.—Effect of Thomas heels on weight bearing of flat feet.

leaving four reporting for what seemed to be typical foot-strain. Of these four, two had an abnormal muscle balance discovered at the first examination and had ignored the advice then given; one later was obliged to give up training on account of persistent rheumatic trouble, and the remaining one had just returned to duty after a long period of comparative inactivity, and was wearing an especially vicious shoe. Six cases have been treated with plates. We can find records of only two cases who were obliged to be off duty for simple foot-strain. There were only two cases of the 133 who were reported later in their course for foot symptoms who showed at their initial examination normal muscle balance. One of these, as has been mentioned above, subsequently gave up her training on account of rheumatism and the other improved after giving up her deforming shoes.

"These statistics would suggest that perhaps among such classes of workers an opportunity is afforded to employ preventive medicine. This preventive medicine is not less important because it deals with a comparatively humble structure, however, which may upon occasion announce its abuse in no uncertain voice."

In more severe cases, and especially in small children or older people, this attempt to control the position of the heel is carried still further by means of the outside iron, as also illustrated here (Figs. 289, 290). In those cases where there is pronounced weakness of the ankle and weakness of the foot, this outside iron with an inside ankle strap and with a Thomas heel built on the shoe gives a combination which very thoroughy controls the tendency to deformity, and which very promptly relieves the disability. We have had several instances in which total diability was changed in a few days to an ability on the part of the patient to go right ahead with heavy work on the feet.

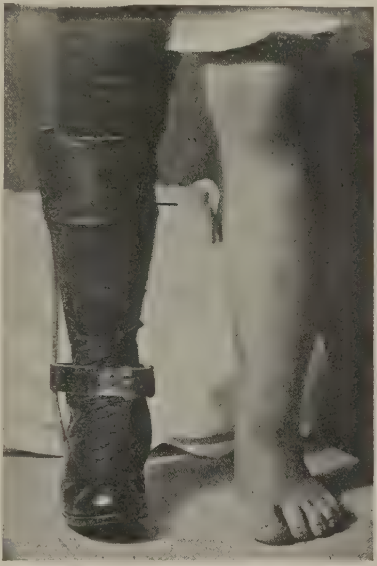
Fig. 289.—Patient in shoe with Thomas heel, outside iron and inside T strap, front view.

In the case of children especially, and in the absence of fixed deformity, it is important to maintain this control of position with as little interference with function otherwise as possible.

Nurse ——— was seen at one of our Lincoln Hospitals in 1920. She was completely disabled for nursing on account of pronated and painful feet. After only a few weeks of duty she had about decided to give up nursing and seek some other occupation. The condition of her feet was severe but early and acute. Without any preliminary treatment in this case, special shoes, outside irons, and Thomas heels were put on. Within two weeks she was doing full duty and she has lost no time from nursing on account of her feet since that time. She is now able to wear any kind of shoe though she usually wears a straight last shoe with Thomas heel for all day nursing duty.

If pronation or flat-foot has become so severe that the feet are swollen and tender, and special heels or even outside irons cannot be worn because of the soreness of the feet and ankles, we then resort to a method of treatment which always applies to any infected or painful joint wherever it is found. In such cases the feet are first carefully placed in the correct position; that is, with the foot fully dorsiflexed and slightly inverted. Forcible correction to this position with the patient under anesthetic and with the Thomas wrench is necessary and justifiable in severe cases and in rigid flat-foot. Then plaster casts are put on (Figs. 291, 292) and the feet are kept completely at rest (immobilized) for some three to six weeks. After the casts are removed, the patient is fixed up with special shoes, outside irons, etc., and can go ahead about his business with comfort.

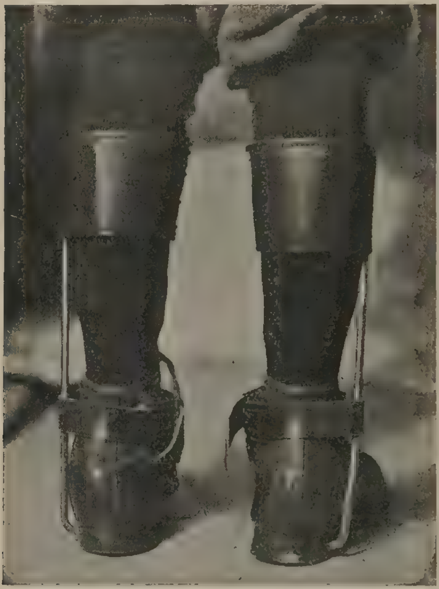
Fig. 290.—Same as Fig. 289, back view.

In cases like the last referred to in which there is also severe cramping or muscle spasm (or even fixed contracture) of the peroneal muscles (the cords of which pass down behind the outer ankle) tendon lengthening by a minor surgical operation (this may be done under local anesthetic) is of great assistance in putting the foot in correct position. After a period of cast wearing one proceeds as before.

Another surgical procedure that may now be said to have established its usefulness in the treatment of weak, badly pronated feet is that of astragalo scaphoid arthrodesis. In this procedure the articular surfaces of the astragalus and scaphoid are removed, the foot well inverted and slightly plantar flexed, and healing is secured in plaster of paris. Doctor Soule of New York has added the use of a bone pin to this procedure. A stable foot with a good arch is thus secured and a number of our patients are entirely relieved following this procedure.

Finally, one must say that there is a definite field for the use of the properly made and carefully fitted "arch support." One of the best of these is that of Doctor Whitman of New York. The exact method of making and employing this support is described by Dr. Royal Whitman as follows:

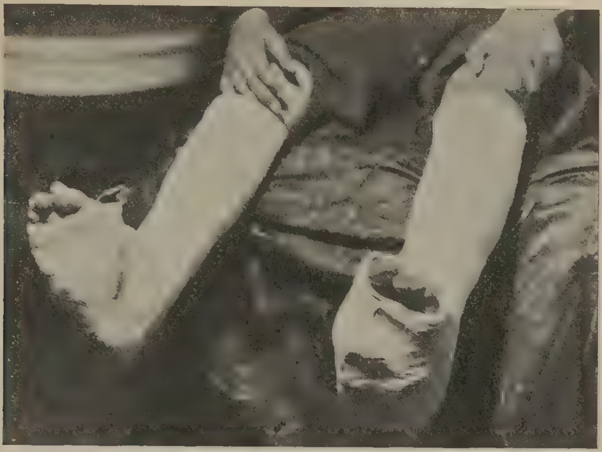

FIG. 291.—Plaster casts, feet in position of inversion and dorsi flexion after forcible correction of heel.

"For the adjustment of such a brace an accurate plaster model of the foot in the proper attitude is essential. This is made in the following manner: The patient is seated in a chair; before him is placed another of somewhat less height, on which is placed a layer of cotton batting covered with a square of cotton cloth. Upon this the plaster mixture is poured. The foot, previously dusted with talcum powder, is then placed in the plaster, resting upon the outer border, slightly plantar flexed with the sole level, and perpendicular to the chair It is an advantage to rest the foot on an inclined plane, the higher side toward the front of the foot, which, together with the weight of the limb, will usually assure slight adduction. For this purpose I usually use a low rocking-chair, which may be adjusted at the proper angle. By raising the margins of the cloth the plaster is

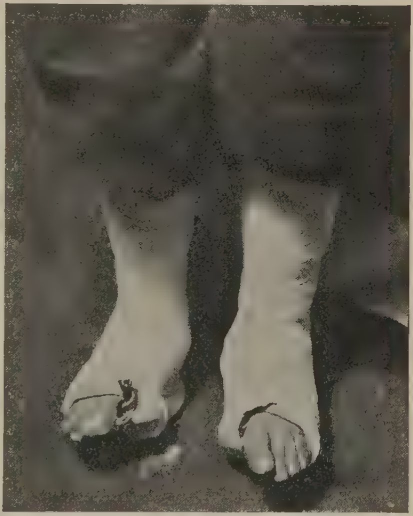

FIG. 292.—Patient standing in casts day following forcible correction.

pressed against the sides of the foot to cover about two-thirds of its extent. The upper surface is then coated with vaselin, and the remainder of the foot is coated with the plaster cream. When this is firm the halves are separated, the foot is removed, and the plaster shell bound together and filled. When the model is prepared certain modifications may be made with advantage. For example: The arch is usually deepened somewhat, particularly at its posterior third, the object being to support the anterior extremity of the os calcis. If the outer border of the cast is flattened a little plaster may be added to round it sightly like the outer border of the hand, so that the brace may not press uncomfortably. If a general bulging on the inner side appears on the cast, indicating residual abduction, it may be reduced somewhat by scraping away the plaster, but if individual bones are prominent, particularly the navicular or the cuneiform, they should be marked with a pencil to indicate to the mechanic that depressions in the brace must be made for them in order that uncomfortable pressure may be avoided.

"On the cast the lines for the brace are then drawn. The inner margin rises above the astragolo-navicular junction. The outer covers the anterior two-thirds of the os calcis and the cuboid bones enclosing this joint. The sole part of the brace extends from the bearing surface of the heel to the bearing surface of the first metatarsal bone. The front and outer two-thirds of the sole are not supported unless there are distinct indications for it, such as breaking down of the metatarsal arch. The brace is made of sheet steel, 19 to 20 gauge, fitted accurately, tempered, polished, and nickel-plated, or galvanized. It is simply placed in the shoe, and, as it is fitted to the foot, it finds its proper place and holds it.

"This brace differs entirely in its principle from those in ordinary use. The foot is placed, as it were, in a saddle, which prevents lateral spreading and the bulging on the inner side that characterizes all grades of deformity. This positive effect is increased by the slight leverage action, for the patient wearing the adjusted shoe, and walking in the proper attitude, the outer flange first bears weight, is pressed down, and in consequence pushes the inner flange slightly against the foot at the movement of greatest strain. The sole part of the brace supports the foot, but the principal pressure often shown by thickening of the skin will be on the inner side of the foot, and there is an entire absence of the hollowed-out, atrophied appearance of the sole, which is so often present when simple, direct support is used.

"The comfort and efficiency of the brace depends, of course, on the accuracy of its adjustment. Thus, if a part of the margin indents the foot it may be turned away with a wrench, and, on the other hand, if the inner flange does not clasp the foot closely, it should be turned slightly inward.

"The brace may be modified in shape somewhat for special cases, and as the appearance and function of the foot improve under treatment, new casts should be made for more accurate adjustment."

One of the lessons of the World War was that in most cases of young robust patients with recent foot-strain, a cure can be brought about by correction of the shoes and a definite course of reconstructive foot exercises. For this demonstration we are greatly indebted to Col. Joel E. Goldthwait of Boston. He conceived and established special training battalions for the retraining and restoration to duty of men whose feet had broken down. To obtain such results, however, there must be a real coöperation between patient and surgeon, and real recognition and treatment of foot ailments.

These measures have all been used by us many times and it is very rare indeed to find a patient who is not entirely relieved by one of these methods, or by a combination of several. The choice as to which treatment is to be used must of course be decided as indicated in Section V, after a careful consideration of the patient, not only with reference to his feet but with reference to all the other points referred to.

The following are exercises of a sort that may be employed for these patients:

1. Patient sitting on a chair. Remove shoes and stockings. Rest feet on the floor with feet dorsiflexed and slightly inverted so that the outer borders of the feet rest easily on the floor. (If the patient stands or walks without shoes at any time he should always assume this position.)
2. Keeping the feet in this position rise from the chair and walk 10 to 20 short steps forward. Then walk directly backward and sit down.
3. After resting for a moment, rise and take 10 to 20 steps to the right and back in the same way.
4. Same exercise walking to the left.
5. While sitting keep the feet in the same position but raise (dorsiflex) the foot until only the outer border of the heel is resting on the floor. Then rise and repeat exercise No. 2 walking only on the outer border of the heel.
6. Similar to No. 3 but on heels only.
7. Similar to No. 4 but on heels only.
8. A final exercise that is especially useful for children is to give them two cigar boxes and about a dozen marbles. The marbles are to be moved from one box to the other—one at a time—from the right-hand box to the left by the toes of the right foot and from the left hand box to the right by the left foot.

Finally, the feet are to be sponged with lukewarm or cool water and vigorously rubbed with a coarse towel until the circulation is thoroughly stimulated. In more advanced cases, dipping the feet quickly back and forth from cold to hot water (contrast bathing) is very useful. This may be kept up for five minutes. Finish with the feet for about one-half minute in cold water and then rub vigorously until they are pink and warm.

A condition commonly associated with weak foot, but which may exist separately, and which is very rarely properly treated and relieved by shoe fitters, or others unfamiliar with the anatomy and physiology of the foot, is what is known as *metatarsalgia* or *Morton's disease* of the foot. This condition is brought about by weakness which affects particularly the front (the transverse arch) of the foot, and which by rubbing together of the heads of the metatarsal bones in the front of the foot, or in some other way, produces a neuritis or neuralgia of the third, fourth, or fifth branches of the nerves that run forward to the toes.

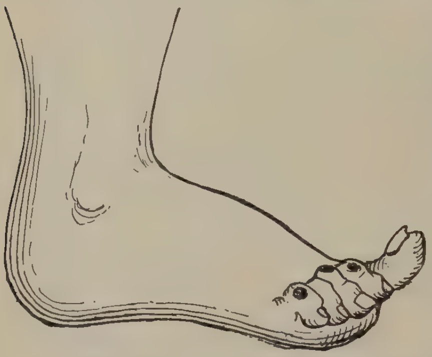

Fig. 293.—Flat transverse arch with contracted, or claw toes.

As a rule we have a flattening of the arch of the front of the foot (transverse arch), but (Fig. 293) sometimes there is no change in the arch that is apparent. Sometimes also we have callouses that form under the heads of these metatarsal bones (Fig. 8), but this is not present constantly. The condition is a very painful one, in fact it is sometimes so severe, coming on as a severe cramp or sharp pain in the foot, that the patient can only be relieved by sitting down and taking off the shoe until the pain passes off. The pain may come on in the same way at night with a sharp, lancinating pain, which is only relieved by changing the position of the bones in the front of the foot, and sometimes only by a period of massage.

This trouble is brought about almost invariably by wearing shoes that are short, or narrow in front, or by having heels that are too high, or by a combination of all these things. The treatment must consist of a correction of these conditions. That is to say, shoes that are wide in front with a straight last and with a good length and a medium heel are necessary. It is also important to see that stockings are not small or too short in the foot.

Sometimes it is also necessary to use inside plates or pads to lift up the bones of the transverse arch so that the irritated nerve is entirely relieved from pressure until healing of the nerve takes place. One of our resorts in the Military Service was the use of what was called the anterior heel, or the sole bar, which is illustrated herewith (Fig. 294).

Surgical removal of the head of the fourth metatarsal has been practiced in some severe cases and is justifiable if there is definite bony enlargement (Figs. 295, and 296). Such surgical treatment must be followed by very careful treatment in the way of pads, braces or sole bars until sound healing has taken place. It is the writers' custom to keep a foot of this sort which has been treated surgically in plaster of paris until it is fully healed. This operation usually gives a painless comfortable foot on which to begin walking when the treatment is finished. It is important to emphasize that in this as in other similar cases *a careful diagnosis must be reached and the type of treatment selected which applies to the particular case at hand.* It is because this point is so frequently neglected that these patients drift from one shoe dealer, and from one chiropodist, to another without getting relief more often than they do.

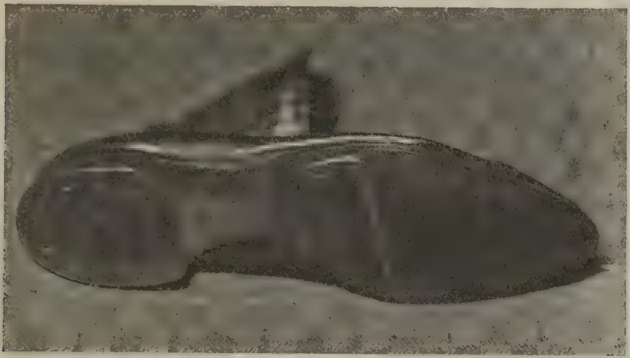

FIG. 294.—Sole bar on shoe.

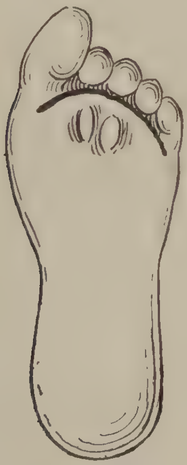

FIG. 295.—Incision for resection of heads of metatarsals. The incision may be modified in length for resection of one or more metatarsal heads. (Modified Hoffman's operation.)

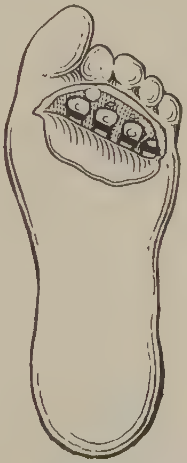

FIG. 296.—Flap retracted showing where heads of metatarsals are removed.

Bunions.—One of the common and most distressing of the painful foot conditions is *bunions* (hallux valgus) (Fig. 258). Bunions are easily prevented. The prevention is simply a matter of wearing straight last shoes of good length, and with plenty of room for the toes. No one who has done this will develop a bunion. Moreover, observance of this rule will correct most of the early bunion cases. After a bunion is fully developed, however, there is practically no recourse except to surgical treatment. Removal of the enlarged bony head of the first metatarsal is necessary before even a proper shoe can be worn. Of course special shoes may be built or stretched into a suitable shape for a person with bunions to wear, but, even then, in time, it becomes an uncomfortable foot in spite of all late attempts to protect it.

An important point to bear in mind is that a painful bunion, even if only in the beginning, is a frequent cause of other foot weakness. A patient continually favors a sensitive toe by standing and walking in unnatural positions; this results in time in foot, ankle and leg strain, and other conditions like metatarsalgia, weak foot, corns, etc., which come along as a consequence.

There has been a good deal of misunderstanding in regard to the surgical treatment of bunions. The operation, if done by someone familar with the proper technique of the operation, and by one who is a competent and skillful surgeon otherwise, is a rather simple matter and one that gives prompt relief.

The operation can be done under local anesthetic so that there is no occasion to hesitate because of the necessity of taking ether. Moreover, the operation wound is usually healed in about 10 days, and the patient may begin to walk in two or three weeks after the operation. There are some who hesitate to have the operation because of the fear of having a stiff toe afterwards, but with the method which is now commonly employed in these cases, namely, the removal of the front end (distal end) of the first metatarsal bone, there is practically no danger of such stiffness.

By this method the end of the bone where the head is cut off is covered with fascia (Figs. 278–280), and the joint otherwise left undisturbed so that there is every possibility for the wound to heal over without stiffening the joint and with the painful, bony enlargement removed and entirely out of the way. Attention must sometimes be given to the removal of the painful bursa or the sesamoid bones under the base of the great toe if that is necessary.

Attention must, of course, also be given to the wearing of suitable shoes, protection of the anterior or transverse arch of the foot, and the prevention of pressure over the operation scar for a time. Subsequently, however, the foot becomes an entirely comfortable foot on which almost any kind of a shoe can be worn.

Another surgical procedure that may occasionally be used is that of osteotomy (division of the bone alone) for correction of the deformity. This may be done much more simply and it is occasionally sufficient.

Amputation of the toe, especially at the second joint, is *never* a proper procedure.

Corns.—Almost exactly the same remarks as have been made in regard to bunions apply to corns. *Corns* of whatever kind and wherever located are due to an undue pressure between the bones of the foot and the shoe. The area of skin pressed upon does one of two things. It becomes callous and hard forming a painful sensitive area on the skin which has to be trimmed occasionally, and which is the kind of corn usually meant when a corn is spoken of. We may, however, have between the toes a different variety, which is a soft corn, but which is caused in exactly the same way. To secure relief of either one, pressure must be entirely relieved, either by the adoption of suitable shoes, or if it is too late for that by the removal of the bones inside the foot, which are causing the pressure. This is important in the treatment of either hard or soft corns, above or inside the little toe. In the case of the corn on the top of the toe, the enlarged portions of bone inside the toe must be removed, and in other corns the enlarged and sensitive head of the metatarsal, which presses against the inner side of the little toe. Such toes should never be amputated except for severe and chronic bone infection (osteomyelitis).

Foot Disabilities Caused by Paralysis.—Finally, we have an entire group of conditions causing painful and seriously disabling conditions of the feet which result from *paralysis* (infantile paralysis or poliomyelitis, nerve injuries, spinal cord diseases, etc.) of the muscles controlling the movements of the leg, ankle, and foot. The conditions resulting from troubles such as the above are of two kinds. First of all, those cases in which there is a considerable loss of power (paralysis) in almost all of the muscles of the leg and foot, and in which we have a severe weakness or flail condition of the entire foot. The second variety is that in which there is paralysis or weakness of one or a few muscles, and in which the deformity and disability are brought about by the contraction or shortening of the remaining strong muscles—which we speak of as *contracture deformity*. The foot may be pulling in any direction and walking may be difficult on account of the weight being thrown on the toes or the heel, or on the inner or outer border of the foot. These conditions have been referred to and need not be described further.

The treatment of all these conditions, whether of flail paralysis or of contracture deformity, must be one of two kinds. Either suitable splints, braces or apparatus must be put on so that the patient can walk with a fairly straight, strong foot and leg, or the deformity must be corrected by an operation, and then later splints and braces applied as may be required. It is a mistake to undertake to correct deformity of this kind by braces alone. This is very rarely possible. It is practically always necessary to correct the deformity either by a bloodless operation (forcible manipulation), and then put on a splint or brace, or second, by a surgical operation to shorten or lengthen tendons or muscles as may be required. It may be necessary to correct the shape or position of the bones that are mis-shapen, out of line, or actually misplaced. Such operations must usually be followed by a period of protection in plaster casts or splints, and then by brace wearing for a suitable period afterwards.

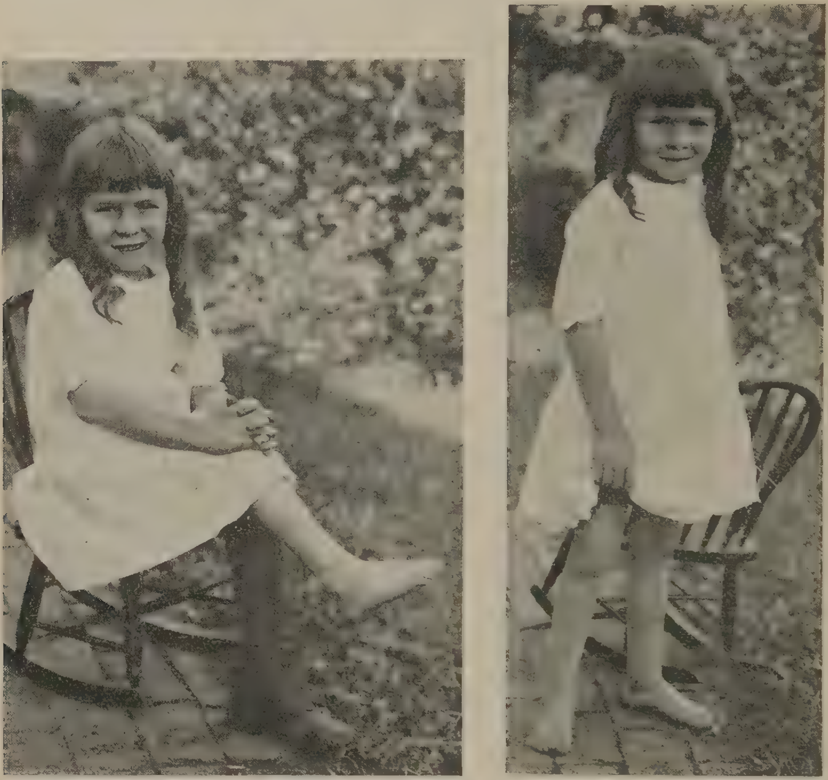

FIG. 297.—These two illustrations are of a little girl who had a flail "drop foot" and calcaneo cavus deformity following infantile paralysis. The operation devised by Dr. Whitman of New York (removal of the astragalus and arthrodesis of the ankle) had been done about eight months before these pictures were taken. The result is a straight strong foot on which she walks without a splint or brace and with no disability or discomfort whatever. (Patient of Dr. Archerd of Clay Center, Nebraska.)

Some very ingenious surgical operations have been devised for the relief of the more serious conditions of this sort. Dr. Royal Whitman worked out an operation some years ago, which bears his name, and which is designed to relieve one of the more complicated paralytic conditions above referred to. This deformity is that of paralytic calcaneo cavus. The operation consists of the removal of one of the bones of the foot (the astragalus) and a backward displacement of the foot. Illustrations of a patient for whom this operation has been done are shown here (Fig. 297). This is one of the most satisfactory operations if properly done, and in properly selected cases.

Another similar procedure, and one which is especially adapted to cavus deformity (contracture of the sole of the foot) alone, or equinocavus, is that of Dr. Arthur Steindler. This is the operation of setting forward the attachment of the strong tendons and fasciæ in the bottom of the foot. The attachments are separated from the heel (calcaneum) and the foot straightened out, as illustrated in the two drawings herewith. This operation is also a most satisfactory procedure, and one which in many of our cases has given great protection and relief.

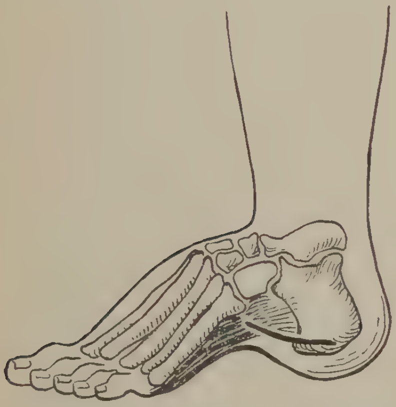

Fig. 298.—Incision for Steindler operation showing contracted tissues under bony structure of foot.

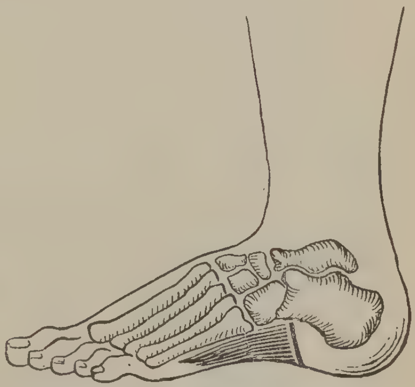

Fig. 299.—Attachments of contracted structures separated from heel, and the foot straightened out.

We have many other surgical procedures like tendon transplantation, and the stiffening of various joints (arthrodesis) about the foot, which are useful in certain carefully selected cases. They are mentioned here principally to emphasize the fact that no matter how disabled or how painful a foot may be, we now have suitable procedures for correction of the deformity, and by which the foot may be made more comfortable in the performance of the functions which every foot should be able to perform.

SHOES AND THEIR FITTING

In considering this subject one should have some understanding concerning the structure and purpose of shoes. Modern shoes are but the evolution of protective devices of ancient origin for the feet—and as our style of dress has changed with the advancement of civilization, so have the rough grass woven sandals become a highly developed foot apparel. Shoes are more than an ornamental adjunct to modern dress. They shield the feet while walking from injury and bruising due to hard or irregular surfaces. They furnish protection against the weather and climatic changes, and have hygienic value in offering a method of keeping the feet clean, or at least preventing flesh contact with the filth of the streets and out of doors, particularly in congested communities.

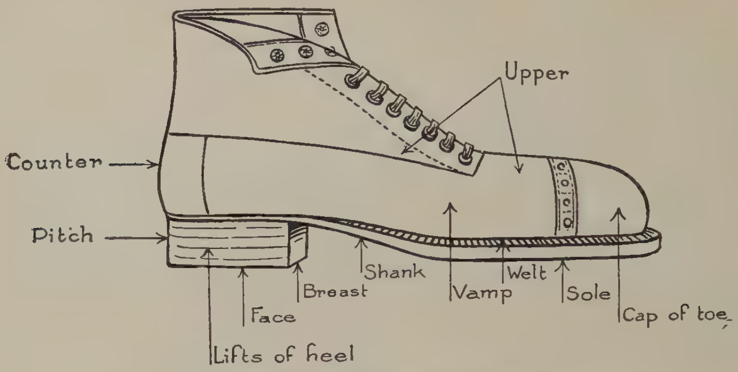

FIG. 300.—Shoe surface marking.

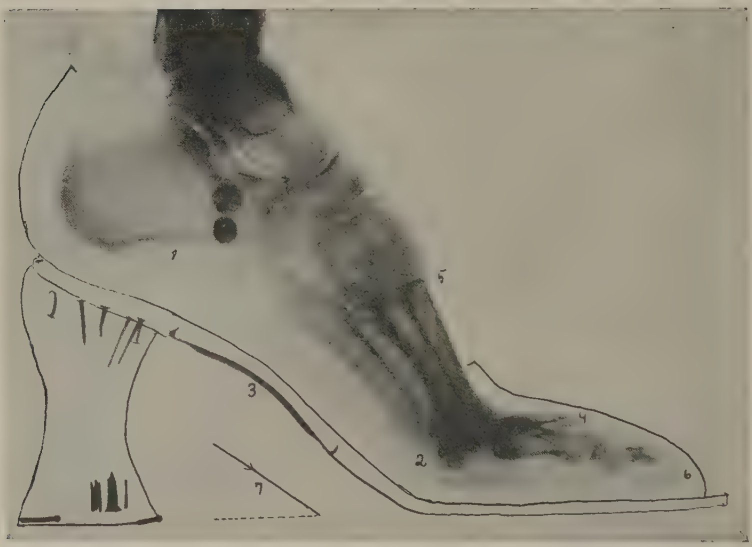

FIG. 301.—Figures 301 to 305 illustrate the effect of the right and wrong type of shoes on the foot. Figure 301, x-ray of the foot of a woman in a high heeled pump. 1, Weight born on anteror portion of os calcis. 2, Weight born on heads of metatarsals. 3, Metal shank. 4, Contraction of toes and turning up of their bases. 5, Forward displacement of anterior or dorsal portion of foot. 6, Toes cramped and shoe too short. 7, Angle at which the foot is standing much as though the patient were on a down hill grade.

Leather, cloth (cotton), and rubber (in its various combinations) are the materials that to a great extent enter into the manufacture of shoes. Of course for dress shoes various silks, laces and other fancy materials are used. Before considering the use of materials one should become familiar with the various parts and surfaces of shoes. Therefore, refer to Fig. 300 for illustration of the various terms used.

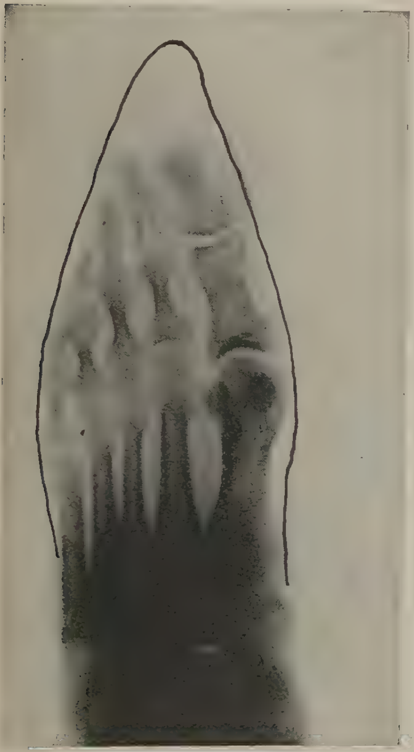

Fig. 302.—Same as Fig. 301 showing anterior portion of shoe deviating outward (pronation), and cramped position of toes.

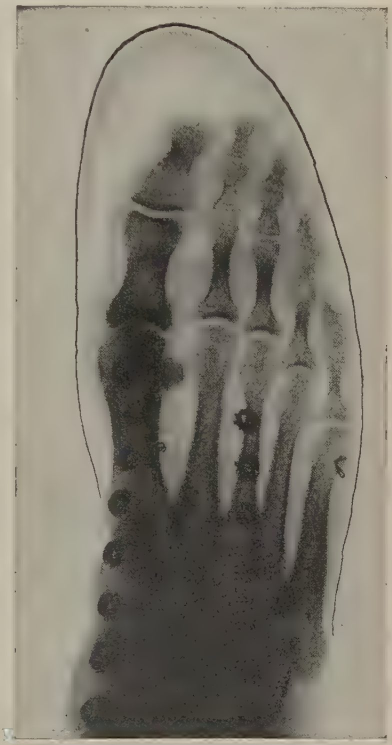

Fig. 303.—Same patient as Figs. 301, 302. Here the foot is in a normal shoe with plenty of room for normal function.

For the sole, heavy cowhide leather of special type is by far the best material. When properly tanned it has a certain amount of flexibility which is of great value particularly in the shank or the part of the shoe bearing the most active portion of the foot while walking. Various rubber compositions, however, are becoming popular and practical for this purpose.

The uppers of shoes are made of almost every kind of leather manufactured with particular reference to the purpose for which the shoes are to be used. The rougher or less refined tough leathers are used for work shoes, and the lighter and finer leathers are used for street wear and dress. With the exception of one particular kind of leather they all have their respective places, and are of particular value. The one exception, the so-called enamel or patent leather, is, with the ill fitting shoe, responsible for most of our minor foot deformities. This leather has no elasticity, no stretching capacity and its cracks and creases as the shoe grows older cause irritation to the feet. Therefore, even a perfect fitting patent leather shoe may cause a tremendous amount of foot disability.

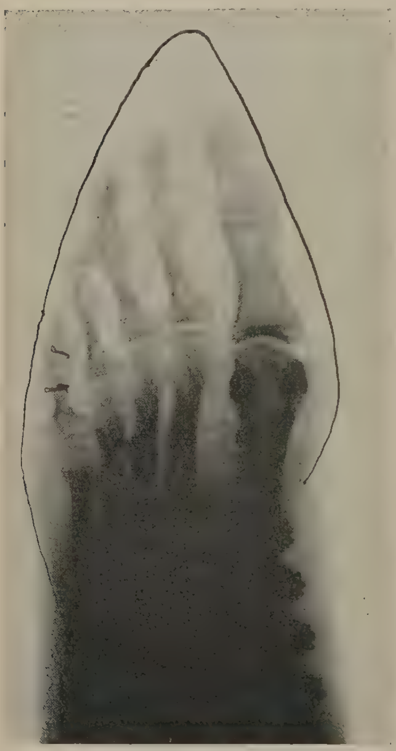

Fig. 304.—The foot of a man in ill fitting and poorly shaped shoe, note the cramping of the toes and the pronation of the anterior portion of the shoe.

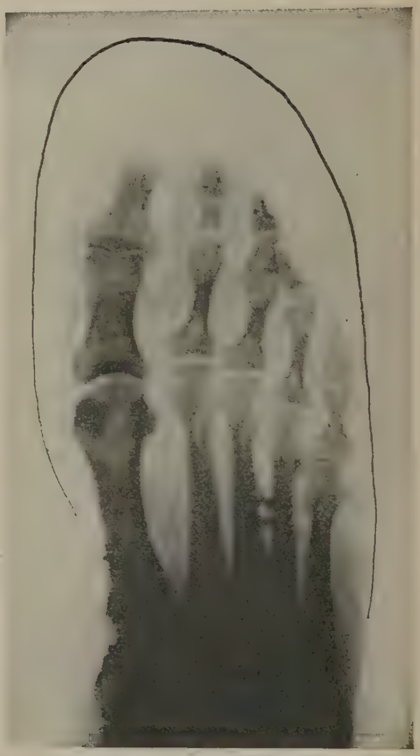

Fig. 305.—Same foot as Fig. 304 in a properly fitting normal shoe.

Canvas or the various heavy woven cloths have practical values in the manufacture of uppers. Canvas shoes are of particular advantage to those engaged in athletics, to persons having sweaty feet, and, when properly fitted, are a source of comfort during the hot summer weather.

Similar fabrics are used for the lining of shoes. This lining takes up the perspiration and furnishes a protection to the leathers that are tanned, dry, and have little or no oily content. Further it gives considerable comfort to the wearer.

Rubber is becoming quite common as a face for the heel in shoes. However, most of the rubber heels are made from too soft a gum and give too much of a cushion for the heel, that is, when the weight is thrown on the heel in the act of taking a step, there is too much of a "drop back" or a "spring down," which is an actual impairment to one who walks a great deal.

With the advance of modern shoe manufacture, standards of lasts have been developed. These lasts are built on wood and are the shape that the inside of the shoe is to be, and they are made to meet certain standard requirements of size. In order to be in style, a great many of these lasts are made without regard for the anatomical structure of the foot, but purely of such a shape that the foot can be made to meet the demands of fashion in shoes.

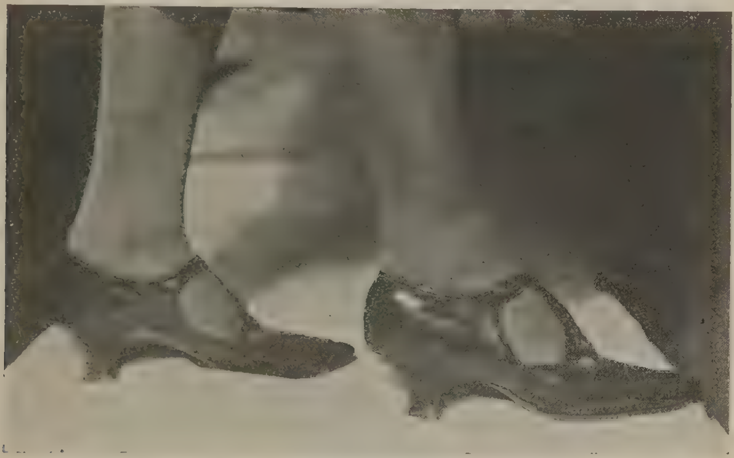

FIG. 306.—Ill fitting high heeled shoes.

The various styles of shoes with their modifications embrace certain fixed principles, many of which are detrimental to the person wearing them, and are actually foot deforming. Among the detrimental features of certain shoes are high heels (Figs. 301–305). They cause the wearer to walk down hill constantly, throwing the weight on the anterior portion of the os calcis, and when combined with pointed toes produce unusual stress and pressure on the ball of the foot or the heads of the metatarsals, and the turning up of the base of the toes, resulting in metatarsalgia, Morton's toes, callouses under heads of metatarsals, hammer toes and bunions. Another bad feature which these shoes have is that the stiff shank, which is usually reenforced with metal, actually prevents the action of the intrinsic muscles of the foot and the tarsal joints while walking, and, though often a comforting contrivance, it is an actual weakening element to the normal activity of the foot. This invention is also used in many low heeled shoes.

Usually the vamp on these shoes and the region across the dorsum of the foot (or where the laces come) is extremely tight, and with the high heel and short pointed toe, a certain amount of upward displacement of the dorsal portion of the foot develops, somewhat similar to that of the previously described toe walking or equinus foot. This may be accompanied by a cavus deformity or abnormally high arching of the foot.

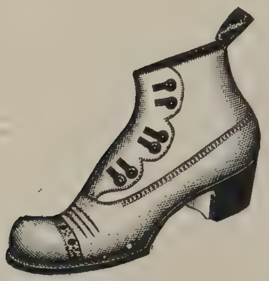

Fig. 307.—Button shoe for a man.

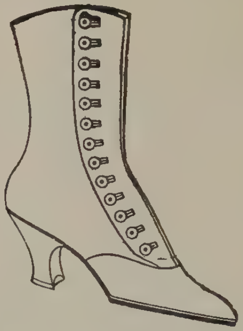

Fig. 308.—Button shoe for a woman.

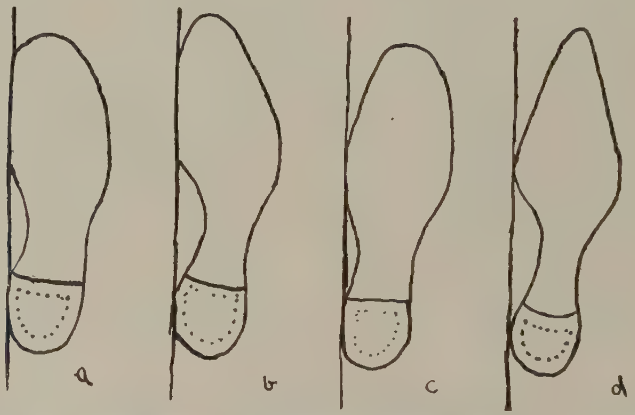

Fig. 309.—(*a*) Normal last. (*b*) Modified normal last. (*c*) Pronated shoe, anterior portion pointing outwards. (*d*) Pronated shoe with pointed toe.

At this juncture it might be well to mention the disadvantages of button shoes (Figs. 307, 308). The buttons are placed at a fixed position and may be perfectly fitting when the shoe is put on, but after walking or being on the feet for some length of time, there comes a slight amount of swelling due to the activity of the foot and the buttons do not allow for any stretching. The circulation, therefore, is impaired and serious damage may possibly come from this one factor.

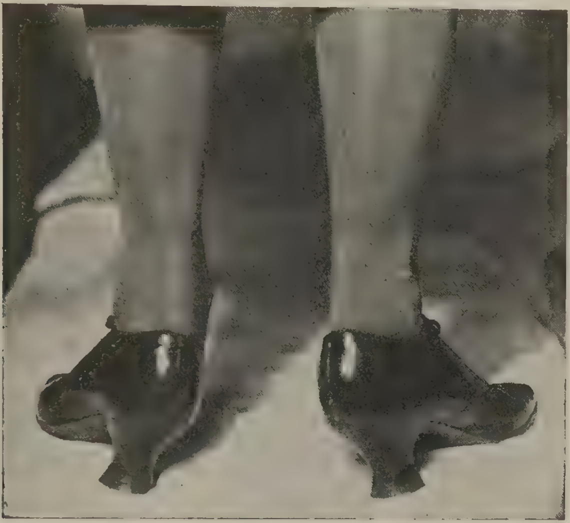

Fig. 310—Patient assuming incorrect standing position because of ill fitting shoes.

There is another style of shoe which may appear to be a relatively good shape, but on close examination the front part of the shoe deviates outward (Fig. 309, *c* and *d*). This feature causes pronation (turning outward) with a resulting weakening of the foot. It was quite a common belief in times past that to walk correctly one must turn the feet out, and it is indeed lamentable that this belief is still common

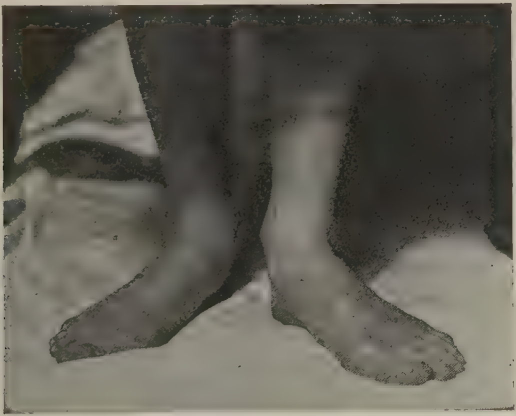

Fig. 311.—Patient having acute foot strain due to standing long periods on ill fitting shoes.

among many of our patients. The shoe manufacturer has taken advantage of this theory and produced shoes that not only point outward but aid the wearer in turning the feet out when walking. Such walking is absolutely against the laws of nature in so far as our method of locomotion is concerned, as it causes foot strain, leg strain, pronation and flat feet (Figs. 310 and 311).

The caps of some shoes are soft while those of others are hard; both are good if enough room is allowed the toes. In the so-called English walking shoe (Fig. 312) the cap is hard and the distance between the top of the cap and the insole is extremely narrow leaving little up and down space for the toes. Such a thin cap often rubs and causes irritation to the toes and may in itself be responsible for corns.

Some shoes have fancy stitching (Fig. 313) which becomes an irritating factor when in close contact with the bony prominences of the foot. Unnecessary seams and stitches should be sufficient reason to condemn a shoe.

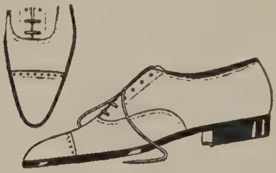

Fig. 312.—English walking shoe with thin cap which often crowds the dorsal or top of the toes causing corns and other disabilities.

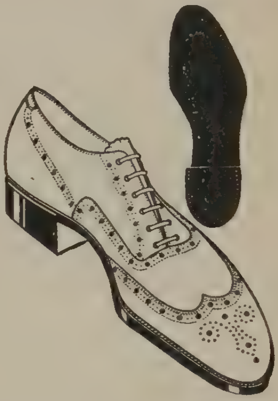

Fig. 313.—Fancy stitching often becomes an irritating factor to the wearer.

These are but a few of the outstanding architectural disadvantages of many of our modern shoes and should be constantly borne in mind and avoided when purchasing a pair of shoes.

It must be remembered that not all feet are alike. There are some short, some long, some thin, and some thick feet—and combinations of these types. One pair of shoes may by ordinary measurements be the correct size, yet upon actual fitting they are not at all proper for the foot. The general impression that a woman's shoe must be of a different shape than a man's is entirely erroneous There are the same number of muscles, ligaments, bones, etc., in a woman's foot as there are in a man's. It is true that a woman's foot may not be as large for her size and weight as a man's foot for the same size and weight, yet they should be the same shape. It is true that many women have worn deforming shoes so long that the feet have to a certain extent taken on an anatomical development similar to the shape of the shoes they have worn. However, even these deformitie do not warrant them in continuing to wear deforming shoes when normally shaped shoes will tend to correct the feet and prevent further weakening. Our experience with the examination and treatment of the feet of millions of men during the past war has aided greatly in appreciating and popularizing a more normally shaped shoe for men and women than has been possible heretofore. Those in service soon realized the suffering caused by ill fitting and badly shaped shoes when they were put to unusual tests on the march and they have been slow to go back to the deforming shoes.

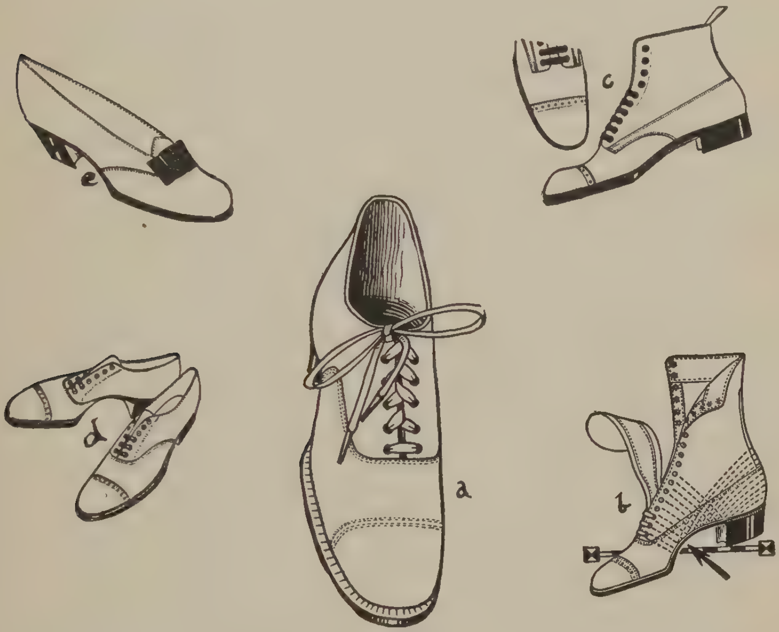

Fig. 314.—*a, b, c, d, e,* shoes following normal contour of the foot.

Certain standards of shoe construction have been developed which make the interior of the shoe similar to the normal contour of the weight-bearing foot. In other words, the last on which a normal or practical shoe is built should be the shape of the foot and should embrace certain fixed principles. First, that a line drawn from the inner side of the heel should extend straight forward (Figs. 309, *a*, *b*) along the inner side of the foot. Second, the shank should be flexible. This is a rather new development in the manufacture of shoes and is accomplished by cutting, first, an especially long upper on the inner side of the shoe and bringing this down under the insole to the outer side of the foot, stitching it firmly in that position, the toe or anterior portion of the shoe being brought slightly inward during this process and the insole sewed directly to this portion of the upper. This permits the longitudinal arch of the foot to rest snugly against this suspension portion of the shoe and prevents the breaking down of the shank.

Third, the fore part of the shoe should follow the normal lines of the foot while bearing weight—with a relatively heavy sole, Fourth, the cap, if hard, should be high enough to give plenty of room for the toes. Fifth, all stitching of the uppers should come where there will be the least irritation to the foot. Sixth, the heel of the shoe should extend out slightly beyond the weight bearing portion of the foot, and be about 1 in. in height with the breast carried well forward on the sole, as the short small heel is a potential weakness to the shank of a shoe and may also be a factor in weakening the feet of the wearer. There should be no tight stitching or compression across the dorsum of the foot, and one should be able to regulate this region by laces.

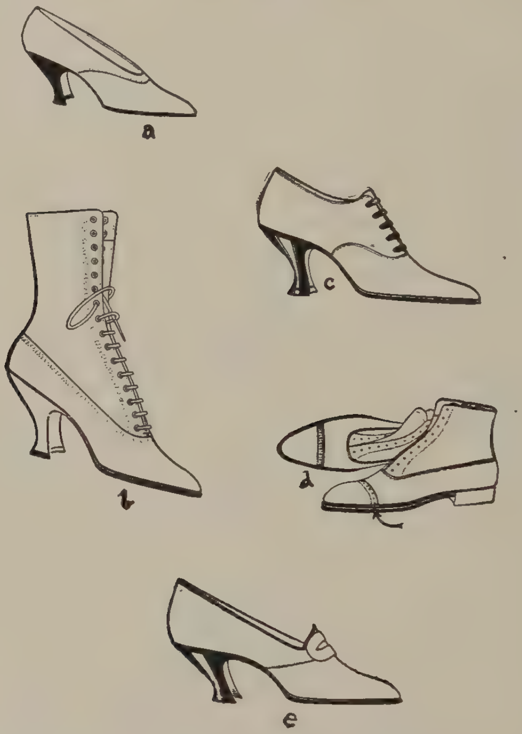

Fig. 315.—*a, b, c, d, e,* shoes of improper shape.

These principles illustrated by the accompanying diagrams (Figs. 314 and 315) are equally applicable for men's and women's shoes. And while the wearing of normal shoes is always advisable for normal feet it cannot be expected, as has been brought out by previous chapters, that they will in themselves, except to a very minor degree, relieve disabilities caused by disease or prolonged wearing of ill-fitting shoes. After proper treatment has been instituted and the feet brought to a normal or as near normal condition as possible, then the normal shoes insure the wearer against future trouble. Furthermore, during that part of the treatment in which the various kinds of apparatus described in the foregoing pages is used, such as Thomas heels, sole bars, wedges, outside irons, and other braces, the normal shoe when combined with these is an aid in maintaining the desired corrective position of the foot during the treatment.

Weakness in the feet of young children is very common and is too frequently encouraged by the usual infant's shoe, although the general shape of the little shoes may give plenty of room. The parents are prone to state that a shoe for a 10 months old child is desired; immediately the salesman selects a pretty little patent leather creation with a white top, four or five buttons, perhaps a dainty blue tassel, and invariably soft paper-like soles. These highly pleasing shoes are brought home and the chubby little feet encased in ill-fitting stockings are maneuvered into them with little regard for the tightness of shoe or buttons. Shortly the little fellow starts walking in these formless shoes. The feet have probably grown since they were procured, yet he is forced to wear them, since he can get into them, and they are so pretty. The child is not accustomed to the act of walking, and immediately with weight-bearing, the foot tends to favor the impediment caused by the ill-fitting shoe and the pronation deformity begins. Every young child should, therefore, be fitted with as great care as an adult. The shoes should be normally formed, have reasonably heavy soles, and uppers of soft pliable leather tightened by laces. They should be changed just as fast as the foot fills the inside space. The idea our parents had that a youngster should wear out his shoes before he had another pair is responsible for much of the foot disability of adulthood today. Once a deformity has been acquired it then becomes necessary to use the corrective devices and measures necessary for the care of the condition.

Of course there are abnormal feet which cannot be fitted except by especially made shoes. These should be made from carefully procured measurements and casts of the feet. And even then they are extremely hard to procure as good shoemakers who are accustomed to doing real artistic work are scarce.

The fitting of shoes involves considerably more knowledge and experience than the ordinary shoe salesman possesses. One should know a good deal concerning the anatomy and physiology of the foot, and all the minor deformities and weakness that can be demonstrated. He should consider the general posture of the individual. He should also take into consideration for what purpose the shoes are to be used, and the kind of work the individual does.

For instance, not only would a very different type of shoe be furnished for one going for a mountain tramp but the size would be considerably larger than that which the same individual would wear as a professional man in his community. Likewise a woman who is continuously riding from place to place in her car could wear a very different type of shoe than one who is walking back and forth to her work. However, these considerations should influence only the type, materials, and size used, and not the style and shape of the shoe as these should always follow the normal anatomical contour of the foot in a well-fitting shoe.

All normally shaped shoes should fit snugly in the heel the heel varying slightly about an inch in height ordinarily. The length of the shoe should extend at least ½ in. beyond the end of the toes while standing. The weight-bearing points of the heads (distal portion) of the metatarsals should rest at a point beyond the flare of the forepart of the shoe just ahead of the shank; in other words, they should rest at the widest portion of the shoe. If they are ahead of this position with the heel in its proper place, the shoe is too short. If they are behind this point, the shoe is too long. The upper across the dorsum of the foot should fit snugly and should not cause the slightest discomfort when the shoe is laced on. Too wide a shoe at this position is often the cause of minor irritations and disturbances of the foot. The vamp should rest snugly against the longitudinal arch.

Quite as essential to the prevention of foot deformity as proper fitting shoes is the proper fitting of hosiery. This factor is often overlooked by those endeavoring to determine the cause of foot disability, while in reality the entire trouble may be due not to ill-fitting shoes, but to improper hosiery. The habitual wearing of short, tight, or pointed toed hose may be responsible for all manner of toe contracture deformities, corns, callouses, and even bunions. One should bear in mind that material and style of hose affect the size, and should buy accordingly, rather than clinging to one number in silk, wool and cotton.

Babies usually wear wool hose or a heavy mixture of wool and cotton. Home knit socks unless very carefully made are to be avoided when a child begins walking, as one very frequently is larger than the other, or the knitting varies in tightness. Most children when beginning to walk are wearing, at least part of the time, the well stretched hose of six, eight or ten months previous. These are far too tight causing a constant binding pressure on the toes, interfering with the circulation and normal activity and development of the foot. These mistakes combined with improper shoes are responsible for much of the early pronation or foot weakness.

As the child gets older, many parents continue to purchase hose by age rather than size of the foot, therefore the stockings are probably either too long or too short. This is inexcusable as the shape of the standard grades of children's hose usually conform to the normal foot and when properly fitted should cause no trouble. When a parent brings a child to us complaining of some foot disability and makes excuses for holes in his stockings by explaining that Johnnie is extremely hard on stockings, we pay particular attention to the fitting of the hose as undue pressure and irritation is responsible for holes. Quite often we find too short or too narrow a stocking or tightly darned or patched portions. Too little care is given to the constant change in size of growing children.

Another bad practice common in large families is the promiscuous passing of stockings from one child to another. When this is done, it should be with a regard to proper fitting. Adults as well as children should purchase hose for a period of time such as six months after assuring themselves of the proper size and material desired. For instance, a patient should determine whether for the six months of colder weather the child would wear all wool, all cotton, or a mixture of cotton wool or silk wool. Then various probable fitting sizes should be taken on approval and tried. If pure cotton is selected, as the child stands it should fill the toe of the hose without stretching and a pinch portion should be felt beyond the ends of the toes. If wool or a wool mixture, the foot should fill the toes of the hose loosely, so that a pinch can be taken on either side of the widest portion of the foot without stretching, and ½ in. should be allowed beyond the end of the toe.

After the size has been thus determined, these stockings should be washed in the accustomed manner, and again tried on. If they still allow full freedom to the feet, a six months supply should be procured and these and only these hose used. It is well to number each pair and as single socks wear out pair the good ones and renumber them in pairs. The fitting process should be repeated at the end of that time. This method assures well fitting hose and will in the long run prove a real economy.

Of course adults should practice with the same care and discretion the purchase of their own hose. Among women's hose, particularly in light-weight silk, there are frequently found pointed and only slightly elastic toes, and irritating seams. Pointed stockings cramp the toes just as does a pointed shoe and become a potentially deforming factor to the foot. The toes of all hose should be elastic, preferably cotton, and should always follow the normal lines of the foot.

CHAPTER CXX

INFANTILE PARALYSIS

(A Problem in Reconstructive Surgery)

By H. Winnett Orr, M.D.

Lincoln, Neb.

One of the outstanding lessons of our recent military experience was that deformity prevention, deformity correction, and the restoration of the patient to usefulness usually involved the application of principles which we had previously employed in the treatment of infantile paralysis. This was almost equally true whether the patient suffered an injury of the nerves, muscles, tendons, bones or joints.

Splinting for the prevention of deformity is nearly always the same. There may be differences of detail in the surgical operation required for these patients. However, immediately following the operative procedures the principles and even the details of mechanical treatment of the affected part for recovery and for ultimate usefulness are always the same. Again, when it comes to massage and other forms of mechanical or occupational therapy, problems and methods differ very little. The reestablishment of patients in their previous employments or their education for new occupations is an obviously similar task in all cases.

It is the conclusion of the writer, therefore, that a careful study of infantile paralysis will yield to any surgeon, methods and expedients for the entire field of reconstructive surgery. The document following is submitted as a contribution to that end.

Definition.—Infantile paralysis, or poliomyelitis, is an acute infectious disease characterized by a destructive inflammation in the anterior horns of the gray matter of the spinal cord and the natural peripheral effects of damage in this region. The disease may appropriately be considered in three stages: The acute stage, the stage of recovery, and the stage of residual paralysis. The characteristic symptom of the acute stage is the loss of power in one or more of the voluntary muscles. This brings a loss of reflexes which may, however, be preceded by a period during which the reflexes are exaggerated. Other symptoms which may be present, but are not constant, are:

Symptoms.—(*a*) Fever, vomiting, constipation, diarrhea, pain—especially along the spine or the course of the principal nerves—stupor, unconsciousness, delirium, convulsions, incontinence of bowels and bladder, retention of urine, dyspnea, dysphagia, or even serious embarrassment of heart and respiratory action terminating fatally.

The paralysis may be, but is not always, preceded or accompanied by a febrile attack. The height and duration of the fever are variable and the severity of the acute attack bears no relation to the extent or severity of the paralysis which follows.

(*b*) Gastro-intestinal symptoms. Many of the patients have attacks of vomiting and other evidences of gastro-intestinal irritation. Both diarrhea and constipation have been reported as common symptoms during the attack.

(*c*) Central nervous system. Before the appearance of paralysis there may be evidence of central nervous system irritaton with delirium, unconsciousness or even convulsions. There are a certain number of patients who in this stage present exaggeration of reflexes and other symptoms of meningitis, encephalitis, or cerebrospinal meningitis and the condition remains in doubt until the characteristic paralysis appears.

(*d*) Genito-urinary system. A number of observers have referred to retention of the urine as an accompaniment of the acute attack. This is probably without very important clinical significance, however. Other kidney and urine symptoms are unusual.

The attacks are frequently, but not always, accompanied by severe pain along the spine, in the extremities or along the nerves which come from the most seriously involved portions of the spinal cord. This pain is increased by pressure or movement and may persist for days or weeks after the acute attack subsides.

A certain number of cases have also been reported in which there are bulbar or cerebral symptoms. There may be paralysis of the face and neck muscles in these cases, or sudden death from embarrassment of the muscles of respiration or heart action. Cases have also been reported with loss of speech, disturbance of vision and difficulty in swallowing.

Diagnosis.—While diagnosis may be a difficult matter up to the time when paralysis appears, the disease may always be recognized immediately upon the appearance of the paralysis. In sporadic cases, however, and in localities where epidemics have not occurred, errors in diagnosis are common. The following is a typical case.

"G. W. Age 7. Attack began Sept. 20, 1913. First physician called pronounced it rheumatism. He treated the patient for six days. Paralysis was recognized by the parents on the second day. The next physician who called pronounced the condition cerebrospinal meningitis. Consultation was called on the eighth day from a neighboring city and the correct diagnosis reached. Complete paralysis existed in both lower and right upper extremities. Massage was instituted on the tenth day, for 30 minutes twice a day. The child screamed with pain at each treatment

"An orthopedic surgeon was now called and on the twelfth day advised discontinuance of massage, made and applied long plaster splints to both lower extremities including the feet. These were bandaged on firmly, to be removed once a day for a warm bath and very gentle friction until all tenderness and pain were gone. The patient was kept recumbent. Four weeks later splints were removed twice a day, more vigorous massage given and the patient was allowed to stand in the splints a few minutes each day."

Immunity.—Natural immunity to infantile paralysis is common. Even in severe epidemics a considerable number of children as well as adults who are exposed escape infection. In one epidemic of more than 1000 cases, studied by the writer, the percentage of definitely exposed persons who escaped was over 50. It is ordinarily stated that the disease is rarely found in adults. But, it is a fact that both in epidemics and when the disease occurs sporadically, infection and paralysis in adults, especially up to the age of 25, are frequently found.

Two attacks in the same individual have seldom been reported. In our own experience there have been two exceptions where there was an apparent lack of immunity as a result of the first attack. One of these is as follows:

"A girl, 12 years old, had the characteristic acute attack beginning August 6, 1909. Symptoms began with temperature of 102 degrees, pulse 130, severe pain in back of head, neck and along the spine. Marked gastro-intestinal disturbance.

"Paralysis began two days later, involving both lower extremities and both upper extremities, more severe in the left arm and the right leg. There was a severe temporary paralysis of the bladder and bowels.

"The patient came under observation in February, 1910. At this time she had made a complete recovery so far as paralysis of the upper extremities was concerned. Her general condition was good, but both lower extremities remained partially paralyzed, the paralysis involving the quadriceps group in both thighs with unequally distributed paralysis of the muscles in both legs. Contracture deformity was already marked and there was inability to stand or walk.

"The patient was anesthetized and the contracture deformity easily corrected by manipulation. Plaster dressings were applied to both lower extremities to prevent a recurrence of the deformity and it was expected that with the aid of crutches the patient would be able to go about in a few days. Six or eight hours after the administration of the anesthetic the patient had not regained consciousness. The temperature had gradually risen to 100 degrees by evening of the same day. In the absence of other symptoms, however, no apprehension was felt until the next day when the temperature continued to rise and the patient became delirious. From this time, the symptoms gradually became more severe. The temperature ran up to 103.5 degrees. More striking evidence, cerebrospinal irritation, began to appear and within 72 hours the patient developed the characteristic convulsions of cerebrospinal inflammation.

"The father of the child, who quite naturally was greatly disturbed by the unexpected consequence of the procedure, refused to permit aspiration of the spinal canal and after death would not allow a post-mortem examination to be made."

All the symptoms, however, and the circumstances under which they occurred, pointed strongly to reinfection with the same organism which caused the original disease. The unfavorable result may fairly be inferred to have been brought about in this way. The writer has not seen this particular accident reported elsewhere and the case is unique in his experience. The same procedure has been followed repeatedly since with other patients crippled in the same epidemic, yet no similar results have been encountered.

Etiology.—Present views regarding the etiology of infantile paralysis have been determined almost entirely by Simon Flexner and his associates at the Rockefeller Institute for Medical Research. Flexner has determined that infantile paralysis can be contracted and transmitted by a presumably specific microörganism which cannot be seen with the microscope, and which passes through the filters ordinarily employed for filtering out the microörganisms.

The mucous membrane of the nose and throat has been shown to harbor the characteristic organism. This is true both for patients who have had the disease and for individuals who have been associated with them. From the patients themselves, it has been possible to isolate the organisms from the tissues of the central nervous system after death. The intestinal mucous membrane is usually found to contain the virus of infantile paralysis, also. The mucous and other discharges, either moist or dry, may be the means of distribution of the disease. The virus has been shown to be quite resistant preserving its virility even after a thorough drying. It has also been shown that mucous membranes continue to have infective properties for a very long time after the other portions of the body have become innocuous.

As has already been pointed out, many individuals seem to possess a natural immunity to the disease. In epidemics, however, a considerable number of persons in the same locality have the disease. In an epidemic observed by the writer, the occurrence of the disease in a large percentage of persons exposed was noted. In one family of 10 children, eight had the disease and two died. In another family of seven children, all had the disease, but only one died. In each of the two families above referred to, several children made recoveries without permanent paralysis.

Pathology.—The specific lesion of infantile paralysis has always been considered to be the congestion, small cell infiltration, and destruction of the large motor cells in the anterior horns of the gray matter of the spinal cord. This has been observed to be most severe in the cervical and lumbar enlargements of the cord, as a usual thing. There is also an amount of swelling of the spinal cord and considerable involvement of the meningeal membranes. The cerebrospinal fluid is of course increased, but is seldom cloudy as in cerebrospinal meningitis. If cloudy, it is so only in the later stages of the disease. Damage to the anterior horn cells either with, or subsequent to, the marked vascular changes which occur are followed by neuritis and degeneration of the nerve roots both anterior and posterior, in the region of the cord most affected. Upon certain occasions there is involvement of the intracranial portions of the central nervous system as well as the cord.

Spastic Paralysis.—The writer has observed several cases which occurred during the epidemic of infantile paralysis, but which presented, upon recovery from the acute attack, a true picture of cerebral spastic paraplegia. These cases are undoubtedly those in which intracranial involvement was more extensive and severe than spinal.

During the acute attack, complications of an inflammatory or congestive type may occur on the part of almost any other portions of the body.

Observed later, the infantile paralysis patients present changes principally in the spinal cord, nerves, muscles, tendons, bones, and joints. There is atrophy and scar formation in the anterior horns of the gray matter of the spinal cord with disappearance of the motor cells and increase of connective tissue. Nerve trunks leading out from these regions in the spinal cord present somewhat similar changes. Muscles or muscle groups in the paralyzed areas largely undergo fatty degeneration. Disuse atrophy of both muscles and tendons occurs. In extremities where there is very extensive paralysis, there is likely to be some failure of bone development, also. If contracture deformities have been permitted to develop, changes in size and shape of the bones, especially at the joints, often occur. Attention has been directed by Doctor Ridlon,* to the fact that overgrowth of the epiphyses, especially at the lower end of the femur, is often a factor in the prevention of suitable correction of contracture deformity at the knee.

Treatment.—Infantile paralysis cannot be classed with the preventable diseases. Adequate care following the acute attack may, however, save a large proportion of those who have the disease from such sequelæ as we now too often see.

This care should begin at once and continue the first year or two after the acute attack. This is the period which we may call the period of spontaneous improvement. Apparently a very few patients entirely recover during the first weeks or months after the attack. A few more show loss of power in certain muscles or muscle groups, but get up and about, and after many months or a year or two of improvement (or compensation) finally appear well again.

The underlying process by which this occurs is as follows: The motor cells in the area of the spinal cord, which has suffered during the inflammatory attack, are partly or wholly destroyed. The muscle or muscles which ordinarily receive impulses from or through this area are "cut off," for a shorter or longer time. Part or all of the damaged motor cells recover. If the peripheral nerve has escaped degeneration the lines are soon "up again." If the damage has been so great that the peripheral nerve must entirely regenerate, it is much longer before impulses begin to be transmitted to the muscle as before.

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* Dr. John Ridlon, Chicago, to whom I am indebted for suggestions and comments also.

In such a case the ultimate effect upon the patient depends upon what has happened to the muscle itself, in the meantime.

If the muscle, whose innervation has been suspended for a time, has been allowed to remain relaxed, overstretched by its unparalyzed opponents, and especially if it has been called upon to undertake its function in weight-bearing without nerve supply, the restored nerve supply later will probably find a muscle or muscle group incapable of responding properly when stimuli again go out over the repaired lines. It is, also, of the greatest importance for us to remember that this whole process of nerve restoration actually occurs much more frequently following infantile paralysis than we formerly thought.

Briefly: (1) The paralysis of infantile paralysis is due to partial or complete destruction of the motor cells and peripheral nerves.

2. This paralysis, so far as the entire nerve supply of any muscle or set of muscles is concerned, is usually temporary. This is so for two reasons: First, because the motor cells for any muscle or muscle group lie at different levels of the cord, and are rarely all involved; and second, because many of those cells actually involved in the inflammatory area recover.

3. Permanent paralysis of any muscle or set of muscles is usually due to the fact that such muscles have been permitted to be overstretched by the contraction of unparalyzed opponents or by unprotected weight-bearing to such an extent that they will not respond to stimuli when the nerve elements regenerate

4. Mechanical protection of the muscles in the affected area against overstretching is the factor of first importance in treatment following the acute attack.*

*Illustration: Case No. 7. "F. T. developed acute anterior poliomyelitis or infantile paralysis the first week in July, 1916, and the writer saw him at his home a few squares from the hospital on the eighth day of his illness. He had a temperature of 101 degrees, a fairly rapid pulse and with the exception of his face and neck muscles, was totally paralyzed. He could not lift either hand or foot from the bed or make any effort to turn himself whatever.

"Dr. ———— and Dr. ———— of ————, in consultation with whom he was seen, at once agreed to his removal to the hospital where he was placed in the quarantine ward on July 10. He remained in quarantine until July 27. During this period he recovered from most of his acute symptoms though he continued to have neuritic pains especially of the lower extremities and was tender upon motion.

"About July 20, plaster splints were put on from the hips to the toes. Relief from irritation was secured and the feet and knees protected against contraction. Upon release from the quarantine ward, new plaster casts were applied. These have been kept on, with occasional changes, ever since.

"During this month (February, 1917) leg braces have been substituted for the casts. No other therapeutic measures have been resorted to. The patient has had the bathing and rubbing which the ordinary hospital patient receives. He has had no massage, no electricity, nor any re-educational exercises, whatever. His treatment has consisted wholly of plaster splint protection and of long confinement in bed. His condition, therefore, presents a particularly interesting picture.

"Today he walks very well and the use of his arms and hands is practically restored. The prospects intimate, that within the two years during which these

Spontaneous Recovery.—Practically all patients who recover during the first six months following the acute attack and many of whom ascribe their recovery to medicines, massage, exercises, electricity, etc., would have recovered just the same if no physician had ever seen them. Why? Because in the relatively short time while nerve restoration was taking place no permanent damage to the muscles occurred.

On the other hand, all those patients who have medicines, massage, electricity, baths and exercises only, and who, after six months or a year have extreme disability or deformity, have been ill-treated. Why? Because, by suitable splints, either in bed or upon undertaking to get about, the muscles temporarily without innervation may be protected against overstretching until such time as the innervation is partly or wholly restored. In this way the first stimuli which do come out by way of the repaired cells and peripheral nerves will find the muscles in condition to respond.

In the order of their importance in contributing to recovery for any patient who has had infantile paralysis are time, splints, exercise, surgery, and braces.

Many muscles, entirely functionless immediately after the acute attack, resume practically normal function before any therapeutic measures are inaugurated. The reason for this is that certain motor cells in the anterior horns of the spinal cord have been only temporarily damaged by the inflammatory process. They resume function, either wholly or in part, before the muscles dependent upon them have materially suffered by the temporarily suspended innervation.

If this innervation is suspended, however, for a length of time which permits profound muscle changes to occur (and this may be only a matter of days or a few weeks) we have then to deal with a problem of so-called residual paralysis.

Deformity.—If it becomes apparent, either at once or later, that the balance between any two opposing muscles, or sets of muscles, is much impaired or entirely destroyed we must then resort to splints. Splints in one form or another must be applied as soon as there is the slightest danger of relaxation or overstretching of paralyzed muscles. This particularly applies to the situation when patients with paralyzed muscles or muscle groups show a disposition early to have their

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patients make progress spontaneously, he will be practically well. There is absolutely no contracture deformity although he still has a few weak muscle groups especially in the lower extremities. Protection by splints or braces will be continued as long as necessary. At the end of two years, if there are any weak muscle groups remaining, recourse may be had to arthrodesis, tendon transference, tendon fixation or whatever else may be necessary. (It is possible to report two and one-half years later that this patient has made an almost perfect recovery. He walks and gets about without appliances of any kind and only the slightest weakness and limp.)"

extremities assume bad positions. This is true of these patients, whether they are lying in bed, sitting, or undertaking to get about.

Patients protected by splints may have their extremities massaged, stimulated by electricity, or make voluntary efforts, any one of which tends to satisfy my third requirement of exercise.

Splints.—Splints are absolutely necessary as a preliminary to and associated with any of these treatments. Any other treatment without the use of splints may be not only inadequate but harmful, because the patient is led to believe that he is being treated, whereas he is being only partially treated. This is not the stage for the application of heavy or complicated braces for walking. Such braces should not be applied at this stage, except for some exceptional reason.

Most of these patients first come under the care of general practitioners. The diagnosis is usually made promptly. When the doctor consults the literature of infantile paralysis therapeutics, it discloses that rest in bed, hydrotherapy, electricity, massage, exercise, splints and braces are all recommended. Splints and jackets are a good deal of trouble. No brace-maker is available. The masseur and the electric battery are convenient and provide something to do. The patient begins to improve—spontaneously—and whatever is being done at the time gets the credit and seems to promise a cure.

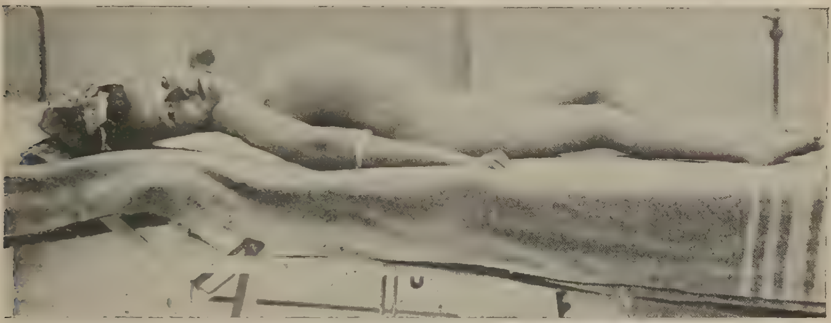

FIG. 316.—An early case of infantile paralysis showing contracture deformities beginning even in recumbency. Note the right drop foot and left external rotation and abduction of the thigh and hip flexion.

The weak muscles, however, are relaxed and beginning to be overstretched, and precious months go by. The patient improves sufficiently so that he demands to be up. Locomotion is permitted, with extremities that are out of balance, or a trunk in which serious potential deformity exists. Even under these circumstances the patient grows stronger, and a kind of improvement persuades those concerned that he is still getting better. As a matter of fact, he is really getting worse. Static and contracture deformities are developing and the patient's condition is changing from one in which deformities may easily be prevented by mechanical means into one in which not even the best of orthopedic surgical procedures will restore what might have been saved by methods of conservation.*

Deformity Prevention.—These observations lead to the following conclusions:

1. It is a serious fault to use electricity, exercises, or massage, or, in fact, almost any other treatment than careful splinting during the inflammatory or painful stage of infantile paralysis. (This may be for six to eight weeks after the acute attack.)

2. No treatment following the acute attack is complete or adequate unless it is accompanied by splints or apparatus to fully protect and relax the muscles affected.

3. It is a serious mistake to put any patient on his feet during the first year after the acute attack unless he is fully protected by splints or apparatus against deformity.

4. The problem of getting the maximum of recovery during the first year after infantile paralysis is about 50 per cent. spontaneous improvement, and somewhere between 25 and 50 per cent. more splinting and apparatus.

5. Finally, orthopedic writers have not succeeded in conveying to the profession at large satisfactory information as to the proper relationship of the different therapeutic agencies appropriate after infantile paralysis.

The means by which the preceding principles are to be satisfied must, of course, vary in the hands of different men. In general, however, I believe that the simple methods are the best. For example: In my own work I have been accustomed to rely very largely upon plaster of paris rather than upon complicated and oftentimes cumbersome apparatus. The attempt to give these patients early apparatus with complicated joints at hip, knee, or ankle, rubber muscles, etc., frequently loads them up with braces which are more of a burden than a help. The simplest device which will enable the patient to get about and which will, at the same time, keep paralyzed muscles relaxed, seems to me to meet every requirement of the situation. In this way, not only do the muscles escape overstretching and remain in the best position, for future usefulness, but nearly, if not quite all, secondary extremity and trunk deformities may be avoided.[*]

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*Illustrative case No. 9. "H. L. was brought to the hospital on October 12 from a neighboring town with acute infantile paralysis. His acute symptoms, except leg pains, had already subsided, although he had been sick only four or five days. The left leg was entirely paralyzed and the reflexes absent. He complained of pain over the entire left extremity and cried out when the limb was moved.

He was kept in the quarantine ward until Nov. 13, 1916. On January 11 his first leg cast was applied, all acute symptoms having by that time subsided. A cast was re-applied February 15. There was still complete loss of power of the tibialis anticus group and to some extent of the quadriceps extensor group. With the cast off there was a decided tendency to drop foot, but on account of the cast having been faithfully worn, no contracture deformity occurred. He stood and walked without difficulty in his cast." This condition is in striking contrast with most of the patients received with tibialis anticus paralysis. Nearly all come in with extreme drop foot and fixed contracture of the Achilles group, so that tenotomy or tenoplasty are necessary before the foot can be brought to a right angle with the leg.

A study of 100 successive badly crippled children who have come to our Hospital revealed the fact that they came, on the average, $5\frac{9}{10}$ years after the acute attack. As to treatment during the period before coming to the hospital the record was as follows: No treatment, 18; medical treatment, 12; osteopathy, 12; electricity, 10; massage, 9; braces, 5; operation, 5; plaster of paris, 1; chiropractic, 1; serum treatment, 1; record indefinite, 26.

To review the treatment briefly, then, one may say that the extensive study and research expended so far upon infantile paralysis has yielded no specific treatment for the prevention, modification or cure of the acute attack. Neither has evidence accumulated as to the efficacy of any particular drug or therapeutic measure. In the light of our present knowledge about all that can be done is to meet the indications in the acute attack as they arise. Fever, gastro-intestinal symptoms and pain may be combated as in any acute infection. It has been claimed by some that urotropin administrations have a measure of specific action on account of their selective action on the central nervous system. Satisfactory proof of this, however, has not been adduced. Counter irritation has been advocated and extensively employed in the form of mustard applications along the spine, hot mustard baths, etc. More active counter irritation has also been recommended and employed, occasionally.

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[*] Illustrative case No. 19. Severe infantile paralysis: Prevention of deformity. "P. B., aged 7, the daughter of a medical man, was seen by Dr. Ridlon in October, 1900. The history of the attack was that on a certain hot day in the preceding August she was out walking in a wood, when she suddenly complained of weakness in her legs, and said she could go no farther. She lay and rested for a while and then seemed to be better and was taken home. The next morning the weakness had passed away. On the following day she went out walking again, and then suddenly fell and was found to be powerless in both legs, and was carried home. On medical examination both lower extremities and the right arm were found helpless. The temperature was 101 degrees and she was in considerable pain. The temperature fell within 24 hours and the pain passed away within three days. She was seen six weeks after the attack and evidence of returning power was noted in the right leg, but the following parts were then paralyzed: The extensors of the right wrist, the dorsiflexors of the right foot, and the whole of the left lower extremity. It was also noted that there was weakness of the left side of the back.

"Means were taken at once to prevent deformity. Daily massage and the interrupted current were prescribed. The right wrist was placed on a malleable iron splint in a hyper-extended position, so as to prevent overstretching of the weak extensors. Posterior splints with the foot pieces at right angles were applied to the legs. It was noteworthy that, even in the short time which had elapsed since the onset of the attack, the left leg had failed to keep pace in growth with its fellow and was ⅜ in. shorter. This shortening has not increased during the two years she has been under observation.

"Recovery has followed rapidly, and the right arm and leg are now perfect. The left leg shows complete return of power in the flexors of the knee, but the extensors are somewhat weak, although still improving. The power of the dorsiflexors is not so great as that of the plantar flexors, so that the foot cannot be dorsiflexed beyond a right angle. There is no deformity anywhere, because the limb has been sufficiently protected."

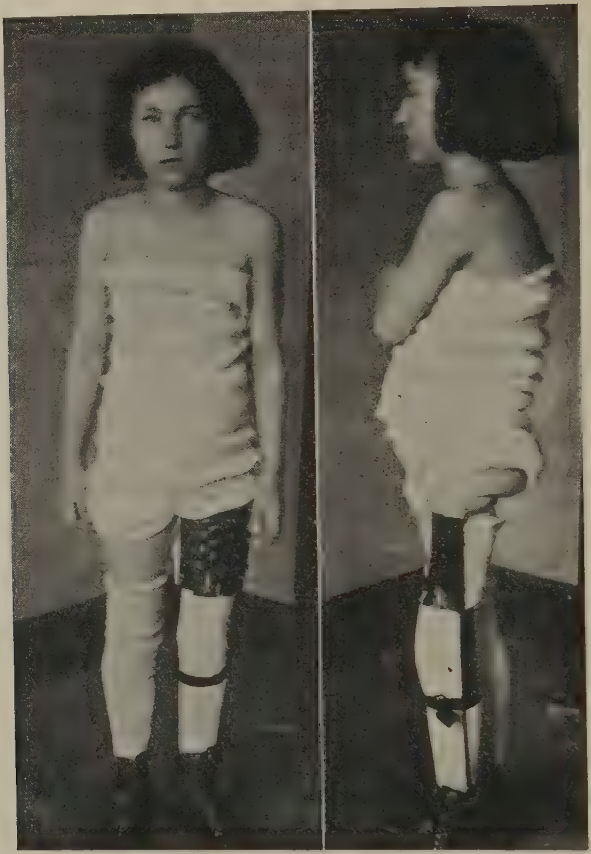

Fig. 317. Fig. 318.

Figs. 317, 318.—Modified walking caliper splint from the conventional splint of the same sort of Hugh Owen Thomas and Sir Robert Jones. In this splint the patient sits upon the padded half ring beneath the tuberosity of the ischium. The double lateral iron extends downward to the heel where it either turns in to the heel giving free ankle movement or is bent forward to the sole of the shoe so as to give right angle foot support. There are many variations of this splint, none of which, however, give better protection to the knee and foot than this simple device. This splint particularly commends itself for the first attempts at walking on account of its lightness and ease of applications. In the early stages of the disease another light splint to maintain the foot at a right angle for the patient to wear at night should be provided. Such a splint may be made of plaster of paris in a few minutes as shown in Fig. 319.

Mechanotherapy.—Hydrotherapy has had its advocates both during the acute attack and later. In the acute attack hot fomentations to the spine with hot baths and ice-bags to the head or back of the neck have been suggested and used. Hydrotherapy in the form of hot baths followed by cold showers or compresses and friction rubs have been suggested for stimulating the circulation in paralyzed parts after the acute attack. These measures have a certain value when intelligently and skillfully employed, but they must be persevered in for some time, to be effective. They must be combined with proper mechanical treatment.

One of the forms of hydrotherapy which has been given a certain vogue by its popularity in the British War Hospitals is that known as the "eau courant or E. C. bath." This bath consists of a special tub in which the water is driven violently in whirlpool fashion, being aerated by a forced current of air at the same time. The affected extremity is submerged and the temperature gradually raised to about 40 to 45°C. In case of nerve injury these baths were regularly employed as a preliminary measure to massage with apparent benefit.

Another expedient was used in the British hospitals and later in this country. It consists of immersing the extremity in melted paraffin in which a much higher temperature can be borne than in water. Immediately upon putting the extremity into the paraffin a cooler layer forms next to the skin. When the extremity is removed from the bath this layer strips off like a glove and apart from the beneficial effect of the heat the extremity is found to be in the most excellent condition for massage.

Electricity in practically every form has been advocated and extensively used for these conditions. It has been used so extensively and with so little discrimination that it may fairly be said to have done these patients, on the whole, more harm than good. Electric stimulation may, however, be so used as to contribute something to the welfare or even the recovery of these patients. The harm done for patients who have been treated with electricity has usually been the result of the fact that the patients were carried along under electric treatment with the impression that measures were being used which would actually restore power to the paralyzed muscles.

False confidence in the efficacy of electricity and neglect of braces or surgical measures, more often than not, leads to conditions worse than the patient would have had if electricity had been left entirely out of the treatment. In the absence of other properly selected treatment of these patients, electricity of and by itself is practically useless.

Associated with proper splinting soon after the acute attack or as a postoperative measure one form of electricity seems to promise more in the way of results than methods previously employed. This is the use of the galvanic current by the so-called Bristow method. Bristow has devised special coils by the employment of which he administers to the affected muscles a kind and amount of stimulation which brings about a graded contraction and relaxation of the muscles very similar to normal muscle activities. The technique of this method is fully described by him in a small book on electrotherapy well worthy of study for its use in infantile paralysis, as well as in nerve injuries.

Massage is one of the best known, but one of the most abused, measures used in connection with the treatment of infantile paralysis. Massage has all too frequently been recommended and its use begun during the acute stage of the disease. The rule should be laid down that massage is never useful and should never be begun until all the tenderness and pain incident to the acute attack have disappeared. Tubby and Jones lay down the rule in italics that to restore to function a paralyzed muscle it should be relaxed to the fullest extent and massaged. This rule if properly interpreted might cover the entire use of massage in infantile paralysis.

Massage by a professional is desirable, but not necessary. We have found it possible in most cases to have a nurse so instruct the mother that intelligent and efficient rubbing, kneading and passive movements both without and with resistance have been given at home. Indeed we have had a little of the attitude toward the professional masseur and especially toward the osteopath that we have toward the prescribing pharmacist. We have found that patients referred to them have been taught to expect too much from the massage and manipulation and are correspondingly inclined to neglect mechanical and surgical procedures necessary to securing the best ultimate results.

Truslow, in a paper several years ago, proposed that a paralyzed extremity should be massaged through openings in a plaster cast. We have not seen the suggestion elsewhere, but have occasionally followed practically this procedure by using a mechanical vibrator through openings in the plaster cast over a relaxed or paralyzed muscle. Exactly how much this contributes to the recovery of paralyzed muscles is difficult to say inasmuch as such muscles recover amazingly if simply relaxed in the plaster cast and supported there with protection against overstretching.

General massage for paralyzed extremities is entirely commendable, but it must be borne in mind that such massage stimulates and assists in the development of the strong muscles as well as the weak ones and unless the proper mechanical measures in the form of splints or braces are used at the same time to prevent contracture deformity, massage may actually contribute to the distortion of the extremity instead of being a benefit to the patient.

Splints are indicated the instant the paralysis appears. The fact that they have so seldom been used at the proper time in patients we have observed has been a matter of serious consequence inasmuch as practically all of these distortions are preventable by proper splinting alone. As soon as the paralyzed muscles begin to relax the strong muscles begin to produce contracture deformity. Indeed, we have seen the belief expressed that the same factors which contribute to the flaccid paralysis stimulate contraction of the paralyzed muscles, thus providing a double tendency toward distortion.

Prolonged recumbency after the acute attack has been repeatedly advocated and is entirely commendable. It is a fact, however, that in some patients who have been in bed only a few weeks after the acute attack, contracture deformity already becomes established and it is almost impossible for patients to stand when they might otherwise be getting up. On the other hand, recumbent patients who are protected by splints against contraction at the knee or hyper-extension of the foot and who in a few weeks show a considerable return of power, are very early able to get about on their feet in proper apparatus without the resultant deformity from weight-bearing which in other patients sometimes produces such disastrous results.

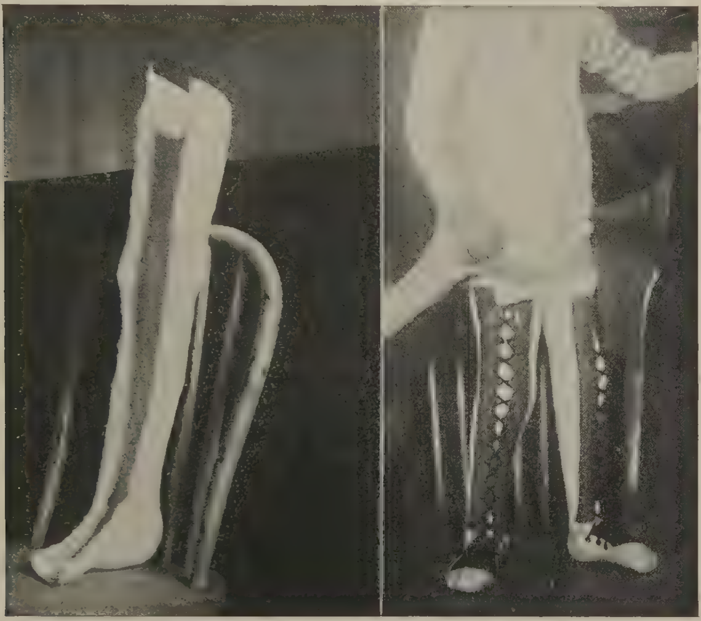

FIG. 319. FIG. 320.

FIGS. 319, 320.—Very light, comfortable splints for both day and night wear may be made by painting successive layers of stockinette, bandages, or other material which has been fitted on to plaster models of the extremities, with celluloid dissolved in acetone. These, when trimmed with leather and fitted with eyelets, lace on very easily and afford very light but very efficient support.

It is important to emphasize to patients the necessity of having these splints worn continuously, night as well as day. In supplying apparatus to patients this point should, also, be borne in mind and such splints provided as will be convenient and comfortable for wearing in bed. In our own work this point has led us into the extensive use of plaster of paris casts. These, if carefully applied with sufficient sheet wadding underneath, make ideal splints. Being unremovable, the patient's exact whereabouts as to his apparatus is always known. With removable splints numerous pretexts lead to a discarding of the apparatus for longer or shorter periods and corresponding intervals of disturbance of the treatment.

The forms of splints used are:

1. Plaster of paris casts (as described).
2. Plaster of paris and celluloid removable splints (Figs. 319, 320).
3. Malleable-iron splints.

These last have as their chief advocate Sir Robert Jones who uses them with great skill and efficiency. Pieces of malleable iron are cut to the desired shape and size, molded by the hands to the position desired and bandaged on.

4. Celluloid—made by painting celluloid dissolved in acetone into layers of stockinette or some similar material stretched upon a model of the part for which the splint is desired. These are trimmed and finished in various ways. One method gives such a splint as is shown in Fig. 320. This splint was entirely made, model, celluloid splint, trimmings and all in our own office without any important apparatus except a factory sewing machine.

5. Leather splints are made by molding wet leather over a similar plaster-of-paris model. The leather is allowed to dry on the model after which it is coated with shellac until very hard and firm. These leather splints are better supported by steel strips and on the whole make rather heavier and less reliable splints than celluloid.

6. Other metal splints may be used. One of the promising materials for the purpose is duralumin which adds malleability and great strength to extreme lightness. A skilled mechanic and plaster of paris models are essential to successful splints of this character.

In several cases we have observed that the early application of splints, especially to the lower extremities, contributes materially to the comfort of the patient, noticeably reducing the amount of pain previously felt especially upon movement. We have also just seen the recommendation of Lange, that plaster jackets be applied to patients in the acute stage for immobilizing the spine. This not only relieves pain according to Lange but contributes to the welfare of the patient otherwise.

Braces are indicated for those patients who under treatment with splints and recumbency have had a sufficient return of power so that exercise and weight-bearing are indicated as the natural means of regaining the greatest use of their paralyzed parts. Contrary to the impression of many, the indication is not to wait for the appearance of deformities before braces are applied. This opinion has been responsible for the serious neglect and gross deformities of many patients. Braces have their chief value either in the maintenance of correction of deformities already obtained or in permitting the parts in normal relationship to resume their natural function of weight-bearing without permitting deformity to occur.

When an extremity can be placed in its normal position inside of a brace, the weight-bearing function can be resumed and all the muscles the weak as well as strong ones are used by the patient to the full extent of his ability without harm being done. This applies, of course, particularly to patients in whom there is paralysis of the lower extremities and in the trunk where deformities, the result of the neglect of this principle, are most frequently to be seen.

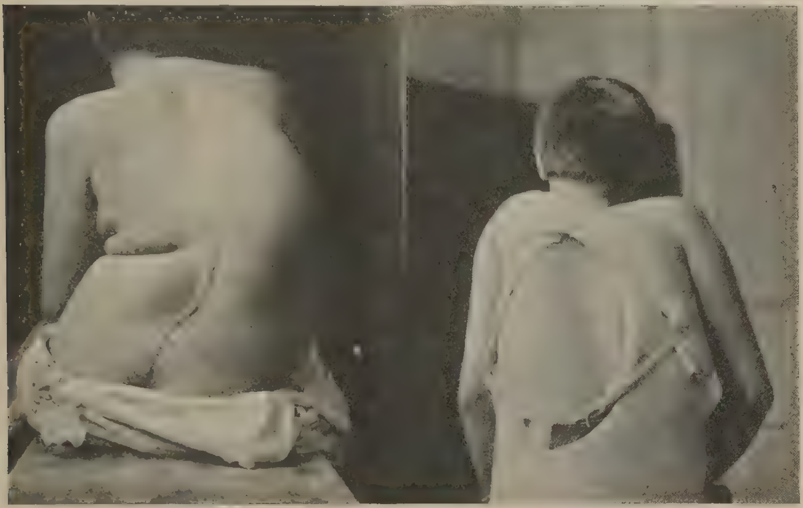

FIG. 321. FIG. 322.

FIGS. 321, 322.—Either closed plaster of paris jackets or removable jackets are more satisfactory for the control of paralytic scoliosis than almost any other mechanical device. Distortions of the trunk have been permitted to develop in cases observed by the writer almost as freely as deformities of the lower extremities. This is doubly unfortunate in as much as extreme weakness of trunk muscles and trunk deformity make it very difficult to put a patient back on his feet even when the lower extremities themselves may be greatly improved by surgical and mechanical treatment.

The adaptation of the brace principle to different conditions and different patients has developed much skill on the part of surgeons and brace makers. A specially made and fitted brace is desirable for each instance.

It may be well to set down here that a common error lies in the supplying of braces too heavy and too complicated for these paralyzed extremities. In general the insertion of joints into braces for paralyzed extremities should only be done in carefully selected cases. Parents and patients have an undue fear of "stiff joints" but the request for ankle and knee joints in many draces for paralytic extremities should be denied until sufficient strength has been acquired so that the patient has full muscular control of such joints.

A point to avoid in the application of all mechanical apparatus is constriction and corresponding interference with circulation in the part. Dr. Henry Ling Taylor has well said that the character of apparatus applied is a fair index of the qualifications and skill of the surgeon.

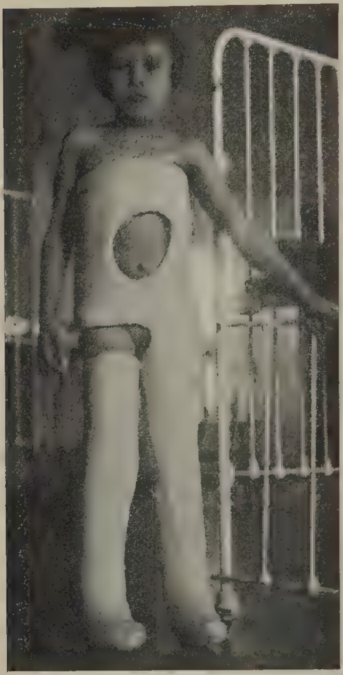

FIG. 323.—This patient illustrates the mode d'emploi for plaster of paris as a temporary expedient in a patient unable to stand or walk. This is a useful expedient for patients in whom rapid return of muscular power is still taking place or in delicate patients in the period before heavier, jointed metal appliances may appropriately be put on. It is sometimes desirable also to employ plaster in this way for the preliminary correction of deformity and to have the patient get about on crutches for a time with the parts or extremities in good position before surgical operation, especially tendon plastic procedures are done.

A considerable number of the patients reported as being completely recovered present upon careful examination a certain amount of loss of muscular power and muscle atrophy.

For a majority of the patients, the second stage of the disease is characterized by a gradual return to function of muscles and muscle groups which were apparently completely paralyzed at the time of the acute attack. Even in those muscles which recover so far as function is concerned, there is usually some atrophy. The period of active recuperation is estimated by various observers at from six months to two years. An interesting observation, however, which has now been made by several of those who have dealt with many of these patients, shows that there is practically no limit to the time when some return of power may be looked for, even in apparently completely paralyzed muscles, under proper treatment. This point will be discussed in detail in a subsequent chapter.

The third stage is in reality difficult to separate from the second, but has been designated as the stage of residual paralysis. For most patients it might almost be called the stage of contracture deformity because it must, unfortunately, be said that a majority of these patients are permitted during the second stage to drift into a condition which leads to unnecessary distortion and avoidable deformity.

Paralysis.—The symptoms of the paralysis of individual muscles and groups of muscles produced by paralysis of their motor nerves are well illustrated in the following schedule.*

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* Quoted.

"Cervical Nerves.

"Paralysis of the anterior division of the third and fourth cervical nerves causes paralysis of the trapezius, as does also paralysis of the spinal accessory, causing a droop of the point of the shoulder and a falling back of the head.

"Posterior Thoracic.

"Paralysis of the serratus magnus causes a downward and backward falling of the vertebral border of the scapula.

"Upper Extremity—Suprascapular Nerve.

"Paralysis of the suprascapular nerve causes paralysis of the supraspinatus and infraspinatus muscles, as shown by the inability to perform external rotation of the humerus, and paralysis of the upper and lower subscapularis interferes with the inward rotation of the humerus through paralysis of the subscapular muscles.

"Circumflex.

"Paralysis of the circumflex causes paralysis of the deltoid and teres minor muscles as shown by a flattening of the shoulder with an exaggerated prominence of the acromion, inability to abduct the arm, and sometimes a sublaxation of the humerus. Paralysis of the brachialis anticus, biceps and supinator longus is often associated.

"Musculospiral.

"Paralysis of the musculospiral causes paralysis of the triceps and all the muscles on the posterior aspect of the forearm, especially the extensors of the wrist and fingers and the supinators, causing "wrist-drop" and inability to supinate the forearm or extend the elbow voluntarily.

"Lower Extremity—Anterior Branches of the Lumbar Nerves.

"Paralysis of the anterior branches of the lumbar nerves causes paralysis of the iliopsoas muscle causing inability to flex the thigh. The leg remains extended if the glutei are intact. This condition is rare except in general paralysis.

"Gluteal.

"Paralysis of the gluteal nerves causes paralysis of the glutei, causing flattening of the buttocks and inability to abduct and circumduct the thigh.

"Anterior Crural.

"Paralysis of the anterior crural nerves causes paralysis of the quadriceps extensor femoris, as shown in the adduction of the leg, flexion of the knee, and inability to extend the knee.

"Anterior Tibial.

"Paralysis of the anterior tibial nerve causes paralysis of the tibialis anticus, extensor proprius pollicis, extensor longus and brevis digitorum, peroneus tertius, causing talipes equinus, toes flexed and dragging on the ground in walking, with hollow sole of foot. If only the tibialis anticus muscle is involved, the foot is in slight valgus.

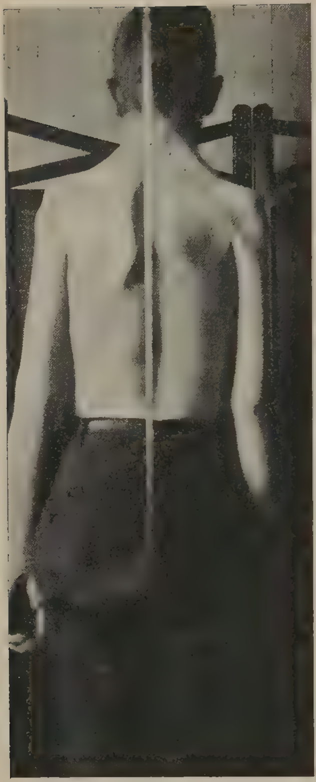

FIG. 324.

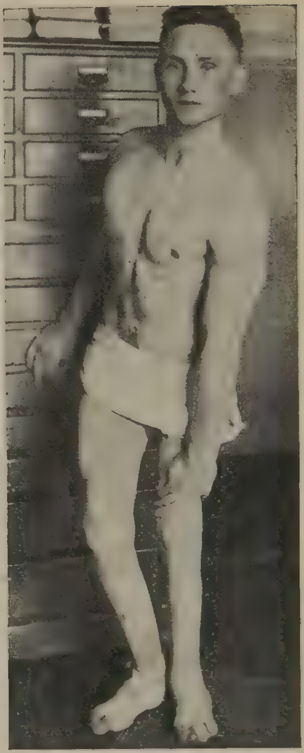

FIG. 325.

FIG. 324.—Extensive paralysis of the shoulder muscles especially of the deltoid gives an appearance like the above. If there is much weakness of the scapular and pectoral muscles such a patient cannot raise the elbow away from the trunk or get his hands up to the face or head. Early treatment in these cases is of the greatest importance. The treatment consists mainly of maintaining the arms in full abduction at the shoulder for at least three to six months after the acute attack. When the patient sits or stands the arm should never be allowed to hang. In this case the weight of the arms has been permitted to cause a downward dislocation of the head of the humerus. In this stage and at the age at which this patient presents himself, arthrodesis of the shoulder is the ideal treatment. Following the operation, plaster of paris should be put on with the upper arm abducted to about 45°, the elbow carried slightly forward and the head of the humerus pressed well upward into the glenoid fossa. The results following this operation are extremely gratifying especially when as in this case the forearm and hand functions have been retained.

FIG. 325.—Patient neglected after infantile paralysis. The left leg is short and the patient can only walk by guarding the knee with his hand to keep it from "breaking" forward. Such conditions are easily prevented by splints or remedied by surgical operation with appropriate after care.

"External Popliteal.

"Paralysis of the external popliteal nerve causes paralysis of the peroneus brevis and the peroneus longus causing talipes varus and pes planus.

"Internal Popliteal and Posterior Tibial.

"Paralysis of the internal popliteal and posterior tibial nerves causes paralysis of the muscles of the calf, tibialis posticus, flexor longus digitorum, flexor longus hallucus, causing talipes calcaneus cavus, inability to plantar flex the ankle joint and adduct the foot, and hyper-extension of the distal phalanges of the toes.

"Dorsal and Lumbar Nerves.

"Paralysis of the posterior division of the dorsal and lumbar nerves, if bilateral, causes paralysis of the extensors of the back causing inability to extend the back, hence a constant maintenance of the body in a position of lordosis and a projection backward of the shoulders. If the paralysis is unilateral, scoliosis is produced. Paralysis of the anterior division of the dorsal and lumbar nerves causes paralysis of the abdominal muscles, causing lordosis without projection backward of the shoulders."

The Field of Surgery.—Surgical procedures are indicated for the relief of fixed deformities, for the stabilizing of flail joints and for the redistribution of power around joints in which the balance of power has been permanently destroyed. These indications of themselves suggest that surgical procedures are as a rule to be resorted to only after other resources have been thoroughly tried or exhausted. It has been generally conceded that most of the return of power to a paralyzed muscle comes in the first six months following the acute attack. As has already been suggested, however, it is now the consensus of opinion of many observers that a return of function is found in many muscles apparently completely paralyzed, even after a year or more, when by mechanical or surgical treatment these muscles are relaxed and afforded an opportunity for the return of function.

Different surgical procedures also are indicated at different periods as will be suggested in the discussion of each.

Forcible correction or bloodless surgery for contracture deformities is usually the first procedure indicated and also one of the best if done properly.

This may often be done manually. The use of the Thomas wrench or some other mechanical device is permissible in the hands of the experienced operator. Stretching must be done so thoroughly that the part can be put up in correct position in splint or plaster without tension. The greatest care must be exercised to avoid external pressure or interference with the blood supply. The latter point requires consideration first from the standpoint of constriction in putting on dressings after manipulation and in avoiding tension on the arteries leading to the part (i.e., the popliteal in correcting knee flexion).

"The natural conclusion from Hibbs' investigations is that the best functional result in paralytic equinus can only be had by restoring the muscle shortened in the development of the deformity to its full length by stretching, not by further shortening it through operative procedure. This is in full accord with the teaching and practice of Newton M. Shaffer, for more than 20 years Chief Surgeon to the New York Orthopedic Hospital and Dispensary, and Hibbs' predecessor in that institution."

This enables the operator, with or without anesthetic as the case may be, to lengthen contracted muscles and relax those which have been paralyzed and afford them an opportunity for return to function.

Correction of contracture deformity by this method, however, should not be employed where enough effort must be used to cause distortion of joints or partial dislocation in other directions. This particularly applies to the knee and ankle where severe fixed contracture often occurs and where open procedure may readily be employed. It is important to remember that a certain amount of over correction must always be obtained and that after such a procedure the parts must be put up in plaster or some other form of fixed dressing and so maintained for a sufficient length of time that recurrence is impossible. It is also to be emphasized that in putting such a corrected extremity in a fixed dressing, it must be done without tension, both on account of the danger of pressure sores and because of the fact that there is great discomfort to the patient unless full correction has been accomplished. This forcible correction may sometimes be accomplished slowly, either by means of special apparatus susceptible of daily adjustment, by means of daily stretching by an experienced operator or in a Zander apparatus or by means of continuous weight and pulley traction. Any one of these procedures usually requires much time of both patient and operator and may be accompanied by considerable more pain and distress than would attend rapid correction with appropriate mechanical treatment afterward.

Tenotomy is sometimes indicated in those cases where fixed deformities cannot be corrected by manipulation. But tenotomy is a procedure of limited utility and one which has frequently been abused. When the tendons are divided subcutaneously, careful adjustment of the over correction must always be made with not only the amount of paralysis and muscles involved in mind, but with an exact knowledge of what is desired in the way of final position of the extremity. The position of the extremity after tenotomy must also be carefully maintained in a fixed dressing for a long time or worse deformities may develop than those for which the patient originally sought relief. Plaster of paris and braces should be worn until there is approximately perfect balance of the corrected extremity and until there is good stability and function of the part under treatment.

After from one to two years of time, splints, and exercise, it may be assumed that all of the recovery reasonable to expect has taken place in the damaged nerve centers. The final effects on muscles and muscle groups may now be determined with accuracy, and recourse may be had to surgery. This may be for the transfer of power from strong muscles to weaker ones, arthrodesis of paralytic joints, or for such other improvement of function as may be brought about by the varied surgical procedures at our command.

It should be emphasized in all our teaching regarding this disease that surgery should seldom, or never, be necessary for the correction of gross contracture deformity, because by proper splinting, such deformities may almost always be prevented (Fig. 323).

In my estimation, the rôle of surgery may best be illustrated by quoting the Whitman operation for paralytic talipes—calcaneo-cavus—as an ideal orthopedic surgical procedure. The operation is recommended for performance at a time when the maximum of so-called spontaneous recovery has been secured, and when, in spite of further splinting and exercise, greater weakness and deformity would occur. This procedure corrects the deformity, further relaxes the paralyzed muscles, and by transferring the tendons of the peronei, contributes strength to the weakened calf muscle. Further, by removal of the articular surfaces of the opposed bones after astragalectomy, stability is restored to the paralyzed ankle joint. Finally, by the application of the brace following the surgical procedure, the extremity is protected as long as necessary, and the brace is laid aside with a previously badly paralyzed and weakened extremity restored to almost normal function.

M. S. was brought to the Nebraska Orthopedic Hospital Dec. 21, 1910. He had been living with grandparents. His mother was dead and the whereabouts of the father unknown. He had had infantile paralysis, was unable to stand or walk. He had made very little progress mentally, and could not even talk well. His delayed progress was apparently due to neglect. This patient's history in the hospital has been interesting because several tendon plastic, bone plastic, and other operations have been done for the relief of deformity and for restoration of balance and strength especially to the paralyzed lower extremities. In 1912, for example, he had tendon transplants of the left hamstring tendons to the quadriceps tendon. Arthrodesis of the left ankle and tenotomy of the shortened flexors and adductors of the left thigh were also done.

In 1914 he had in the right leg a bone graft from the tibia to the lower and outer border of the fibula, the graft extending down into the cuboid. This was done with the idea of affording stability at a right angle. There was also an additional correction by Thomas wrench of deformity of the left foot. In 1915 the feet were again corrected by wrenching and he was again put in a cast. Up to 1917, however, nothing more had been accomplished than to have him occasionally on his feet in plaster of paris or in a body brace, including the entire length of both lower extremities.

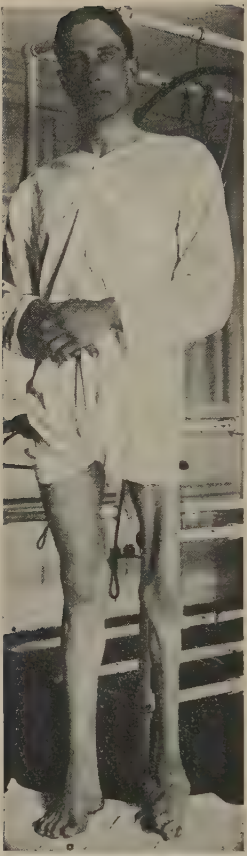

Fig. 326.

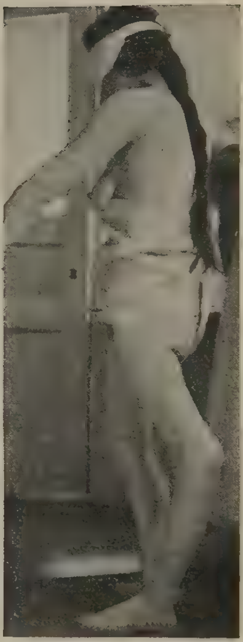

Fig. 327.

Fig. 326.—Adult patient with irregularly distributed paralysis in both lower extremities. The extent of the paralysis is much greater in the left lower extremity below the knee. The tibialis anticus group, the peronei, and to a certain extent the calf muscles are all affected. It is in this condition especially with paralytic calcaneo cavus deformity that the astragalectomy of Dr. Whitman with arthrodesis finds its most successful application. This operation yields for such patients a stable ankle joint with the foot in good position and with a shortening of the extremity of not to exceed one-half to three-fourths of an inch.

Fig. 327.—The above represents exactly the kind of patient for whom arthrodesis of the knee is the desirable procedure. This patient is one for whom various braces and splints had been employed but who had had the usual experience of having braces break, wear out, and become too small, until she finally arrived at the condition shown above. The writer has had a number of cases like the above who have become quite independent of apparatus and who get about comfortably after arthrodesis of the knee and with the knee fixed in about the same angle as that shown above but forward instead of back. The writer has had no instance of failure in these cases from any cause but he has selected his cases carefully with reference to the following points: (1) No attempt has been made to arthrodese the knee in a patient younger than twelve years. (2) Complete union between all the bony structures of the knee including the patella has been obtained. (3) The minimum period of perfect immobilization first in plaster spicas and then in plaster extending to the hip has been four to six months.

On Aug. 22, 1919, he was found not to have made any recent attempts to stand on his feet. He was content to ride about in a little wagon and he had not made good progress either mentally or physically. Arthrodesis of left knee was decided upon and done. The articular surfaces from femur, tibia, and patella in the left joint were removed and he was put in a long plaster. A leg brace for the right leg was provided. This consisted of a double lateral iron with a right angle foot support. With this brace on his right leg and the plaster of paris spica on the left he walked better than he ever had. As soon as the bone formation in the left knee was complete, the plaster spica was laid aside and he walked well with only a light foot brace on his left foot and a long leg brace on the right.

This case illustrates some of the several possibilities that lie in even the most extreme conditions that follow infantile paralysis. Doubtless surgical operations have been done in this case which have not contributed very much to the patient's progress or his present condition. In general, however, it may be said that the prolonged period of hospital care really counts as nothing when it is considered that this patient at the age of 13 has become an ambulatory, independent individual, instead of a wheel chair invalid. His legs are useful for locomotion and his arms and hands if trained faithfully will fit him for skilled work. It is proposed to have him trained in shoemaking which will render him at least by the age of 18 entirely capable of supporting himself or even others who may become dependent upon him.

Tendon lengthening by a tenoplastic operation or by the use of silk strands is frequently to be preferred to simple tenotomy. In this operation also, careful allowance must be made for the amount of correction desired and also for the relaxation of the paralyzed muscles. That is, there must always be a measure of over correction of the deformity but not a sufficient amount to over balance the corrected joint in a direction opposite to that which existed originally.

Especially in cases with fixed contracture deformity of the knee in flexion it is important to do a plastic lengthening of the hamstring tendons rather than to do tenotomy. The writer has found almost as many cases of recurvatum of the knee due to uncontrolled deficiencies of the hamstrings as he has talipes calcaneus and calcaneo cavus due to over lengthening of the tendo Achillis (Figs. 325, 327).

Muscle and tendon transplantation were hailed a few years ago as the coming operations for the relief of deformities and restoration of function in all paralyzed extremities.

"The history of tendon-grafting dates from 1882, when Nicoladoni implanted the central ends of the severed peronei into the tendo Achillis. The immediate results of Nicoladoni's operation were promising yet Maydl states that the anastomosis finally separated, and all the benefits of the operation were forfeited. Tendon transplantation was revived by Drobnik in 1892, and simultaneously and independently by Parrish of New York, in a modified form. Drobnik introduced tendon splitting, inserting a reinforced strip from a normal muscle into a paralyzed tendon. Since 1892, many surgeons have worked in the same field. In this country, Sir F. Eve, R. Jones, Montgomery, E. M. Little, the author and others; in America, Bradford and Lovett, Whitman, Goldthwait, Waterman, Dane and Townsend; in Germany, Hoffa, Vulpius, and Lange; and in France, Kirmisson and other surgeons."*

A full review of the literature of the subject up to 1904 will be found in "Chirurgie Orthopedique" by Berger and Banzet on pp. 250 and 251, in the works of Hoffa, Vulpius, Lange, and Codivilla.

The precedures coming under this head have been extensively used and much discussed, but many of the earlier operations were greatly disappointing.

Tendon and muscle transplantation are done at points where the balance of an extremity has been permanently and seriously disturbed, and with a view to carrying power from the concave to the convex side of the deformity, that is, from the contracted to the relaxed side. As an illustration, in talipes calcaneo valgus, the tendon of the peroneus which is over acting may be partially or wholly attached to the tendo Achilles, as a consequence of which the valgus deformity is relieved and the calcaneum is furnished with new support.

There are five principal methods of transference: (1) The transference of the whole of the tendon of a strong muscle to the whole of a weak one. (2) The transference of the whole of a strong muscle, simply attaching it to the tendon of a weak muscle without division of the latter. (3) The transposition of a tendon so that the strong assumes the entire function of a weak muscle and the weak of the strong. (4) The splitting of the tendon of a strong muscle and the transfer of a portion of the strong muscle to the tendon of a weak muscle. (5) The splitting of both tendons and the transposition of parts of the strong muscle and the weak one.

These procedures originally involved the tendons alone. They have more recently been modified so that it is common now for the tendon of a strong muscle to be transferred in such a way that a periosteal attachment is secured in a new position so that the strong muscle takes upon itself the function of the paralyzed muscle (Jones, Gallie).

Some operators prefer to carry transplanted tendons through holes which have been bored in the bone and securing them by loops which are stitched firmly in place in their new position (Whitman, Jones).

Still others, following the lead of Lange, make use of silk strands to lengthen transplanted tendons which would otherwise be too short for making periosteal or bone attachments. These silk strands have been found to adapt themselves both functionally and histologically to tendon function.

* Quoted.

In the selection of tendons for transference, the effect upon the extremity as a whole must be as carefully kept in mind as the immediate objects to be attained in the region of the paralyzed muscle. In other words, we must carefully avoid the removal of such a quantity of power from the region of the strong muscles as will disproportionately weaken it as compared to the region to which strength is being transplanted. A rigid and accurate technique must be observed in these operations. The principal points to be observed in about the order in which they occur relative to the operative procedure are as follows:

Existing deformity must be entirely corrected and over correction of the deformity provided for. The preparation of the patient and the surroundings must enable the operator to observe rigid asepsis. The extremity to be operated upon should be rendered bloodless. Sufficiently extensive incisions should be employed so that all structures to be dealt with are freely observed. The appearance and character of the muscle when exposed must be taken into account in estimating where strength lies and to what point it must be carried. Redness of the muscle in general indicates healthy tissue and strength. Yellowish muscle in general indicates degeneration and weakness. None of the tissues should be submitted to unnecessary traumatism or injudicious handling. The damage or division of nerves and important vessels should be carefully avoided. In the transplantation of a tendon the aim should be constantly kept in mind of having traction as nearly as possible exactly in the direction in which support is desired. It should not be attempted to make transplanted muscles act around corners or through entangled structures.

In making attachment of tendon to tendon, of tendon to periosteum either directly or by prolongation of the ligament with silk strands, moderate tension only must be employed. Attachment must be made with the part in exactly the position desired both at the time and subsequently in the fixed dressing. Violent manipulation after these attachments must be carefully avoided. Hemorrhage must be carefully controlled or, if not satisfactorily controlled, suitable provision for drainage and subsequent dressings so provided for that secondary infection or disturbance of the part by improper motion will be impossible.

Attention must be called to a principle which must always govern in splinting muscles or muscle groups wherever located. The object should be to put paralyzed muscles or muscle groups in a position of complete relaxation. This is a point which has been sufficiently emphasized by Sir Robert Jones, R. W. Lovett and other authorities on the subject. It has been pointed out by these writers and others that even following operations upon paralyzed extremities in which no hope of recovery of muscle power had been entertained there was after the operation a return of the power of voluntary contraction to muscles which had been paralyzed for a very long time.

Something has already been said as to the importance of aftercare following this and other methods. A paragraph from Lovett, however, crystallizes the whole matter in a way which should impress everyone who has to do with these conditions.

> "To perform a tendon transference, to fix the leg in plaster for some weeks until muscular atrophy has become marked, and then to allow walking unprotected is bad treatment. Unless the operated-on leg is massaged from the sixth week, and the newly transferred muscle is trained to its new function shortly afterward, and unless this massage and training are carried over some weeks, the best results cannot be expected. No matter how good the operation, it must be followed up, or the operation will be regarded by the individual operator as an over-estimated procedure; if, on the other hand, he performs a sound and reasonable operation, and has his patient treated properly over a period of about six months he will find it, in a large proportion of cases, one of the most satisfactory procedures in operative surgery."

In general it may be said that these early failures as well as present failures are due to three principal causes: First, operation in improperly selected cases; second, operation in suitable cases but without adequate consideration of the requirements of the individual patient; third, inadequate after-care.

Under the first head it is to be borne in mind that for this procedure we must have a patient in whom the amount of remaining power will really give a restoration or partial restoration of function to the part if properly redistributed. Under the second head a careful analysis must be made of the amount of paralysis existing in the weak muscles and the amount of power present in the strong muscles and the manner in which the power available can be distributed to replace that which has been lost. In this procedure also, it is important to maintain the operated extremity in a fixed dressing for a considerable time and to carefully supervise the first beginnings of a return to function. Casts and splints or braces should be worn as long as there is any tendency to relapse or the development of deformity in a new direction will be probable.

It is under the third head that many failures attributed to the operation must be classified and also to the after-care must be given much of the credit for the good results which some operators have reported.

> "No. 15.* —Thirteen year old girl. After seven years of paralysis this patient presented the following conditions for treatment: Moderate calcaneus deformity with pes cavus, slight power in all lower leg muscles, tendo Achillis group weak, partial paralysis of quadriceps and biceps muscles in thigh. No brace was worn.
>
> "Tibialis posticus and peroneus tertius tendons were transplanted into tendo Achillis. Transverse osteotomy of os calcis and the elevation of its posterior fragment for reunion in corrected position was performed. Four years and eight months after operation the foot is still in calcaneus and cavus position of less degree. Position in weight-bearing is good but more unstable. There is some strength in tendo Achillis. No brace is worn although one has been tried unsuccessfully. The patient states that she turns her foot more than she did before the operation and the condition, she thinks, is worse. From an anatomical standpoint there has been some improvement in form at the loss of the more important feature, functional stability—an unsuccessful result."

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* A series of cases reported by others have been taken as a basis for comments by the present writer on surgical procedures and after-care.

The above patient presents a surgical problem which affords opportunity for one of the most successful of all the surgical expedients available in infantile paralysis—the Whitman astragalectomy for calcaneo cavus. The exact method of Doctor Whitman is appended.

"The line of incision begins at a point about an inch above the extremity of the external malleolus, midway between it and the tendo Achillis, and is continued downward and forward about three quarters of an inch below the malleolus over the dorsum of the foot to the external surface of the head of the astragalus.

"The sheaths of the peronei tendons are opened and the tendons are cut below the malleolus and drawn backward. The bands of the external lateral and interosseous ligaments are divided and the head of the astragalus is freed from its attachments to the tibia and scaphoid. An elevator is then inserted between it and the os calcis, and foot being forcibly inverted, the head of the astragalus is drawn from the wound, and, the attachments on its inner and posterior borders having been cut or broken, it is removed.

One, then, prepares the new articulations. A thin section of bone is cut from the adjoining surfaces of the cuboid and os calcis. On its inner side a knife is passed about the internal border of the scaphoid and the ligament is separated by an elevator. The foot is then displaced outward and the malleoli are laid bare by dissection from their ligamentous attachments and are reshaped somewhat on their internal surfaces to fit the new articulations. The peronei tendons, freed from the lower extremity of the fibula, are passed through a slit at the base of the tendo Achillis, sewn firmly to it, and then drawn forward and reunited to their distal extremities.

The foot is now displaced backwards, the external malleolus overlapping the calcaneo-cuboid articulation; the internal, the navicular. The wound is then closed with catgut sutures and the foot is fixed by a plaster splint in an attitude of moderate plantar flexion and abduction.

In the routine of hospital practice, the operation is performed under the Esmarch bandage. The tendons are sutured with Lange silk. The wound, having been cleansed with warm saline solution, is closed without drainage. The foot and limb are bandaged with sterilized sheet wadding, over which a light plaster is applied, holding the foot in the attitude described, and the leg at a right angle to the thigh. The limb is afterward suspended between tapes running from the head to the foot of the bed. Great care is taken to avoid constriction. To this, and to the rest assured by the plaster splint, and to suspension, is ascribed the very slight discomfort following the operation, and the absence of complications.

At the end of about three weeks the first support is removed, and the walking plaster splint is substituted, extending to the knee, and fixing the foot in the same attitude of moderate equino valgus, the sole being equalized by the incorporation of a wedge of cork. The patient is encouraged to walk with equal steps, and to bear weight on the forward part of the foot. At the end of from four to six months the new joint will have become stable, and the fixed support may be discarded for a shoe arranged with a cork wedge beneath the heel, of sufficient thickness so that the foot is compensated for slight equinus, and if necessary the outer border of the sole is thickened somewhat to prevent a tendency to inversion."

"No. 6.—Thirteen year old girl. This patient applied for treatment after 10 years of paralysis. There was moderate equinus and moderate varus position. The peroneal group of muscles was weak. There was good power in tibialis anticus and in tendo Achillis group. No apparatus had been worn.

"The operative procedure was as follows: Tibialis anticus tendon transplanted into external cuneiform bone; cuneiform osteotomy of neck of astragalus; lengthening of tendo Achillis. Eight years and one month after operation the foot is slightly under-corrected in weight-bearing position, and slightly more so without weight. The stability is excellent. There is voluntary and passive dorsal flexion of 75 to 80°. No abduction but free adduction was possible. The transplanted tibialis anticus is actively functioning Tendo Achillis group is of moderate strength. A brace is now necessary. The patient would go through the experience again for the benefit derived from the improved position and function of the foot."

In general, operations of this kind prove disappointing. Either one fails to get stability of the ankle joint, or varus and drop foot persist. Two procedures have proven of value: Suspension of the foot by the Jones or Gallie methods or arthrodesis of the ankle joint. With either of the latter procedures brace wearing becomes unnecessary—toe drop is overcome. If there is a tendency toward either varus or valgus with equinus and any considerable loss of dorsiflexion arthrodesis is most promising.

"No. 7.—Eleven year old boy. After many years of paralysis, this boy appeared for treatment. His condition was as follows: There was considerable varus deformity with adduction of the forefoot; also, slight equinus. Patient walked on outer edge of foot and ends of extended toes. An operation had been advised after previous other manipulations. No brace was worn.

"The tibialis anticus tendon was transplanted into the cuneiform osteotomy line of the external cuneiform bone. Five years and one month after the operation the foot is considerably under-corrected. Dorsal flexion is limited passively to slightly less than a right angle. Calluses are still present on the anterior outer part of the foot. There is slight function in the transplanted muscle. No brace is worn. The patient thinks the surgical operation was of some benefit and that there is some improvement in the position of the foot."

The after lesson in this case may be that the patient would have been more improved by an arthrodesis of the ankle. Suspension of the foot by the tibialis anticus tendon (Jones or Gallie methods) might have given a more useful foot. In less aggravated cases the method of Mayer herewith may be applied with good results.

"Conversion of the Tibialis Anticus into an Abductor and Pronator.

"This operation is indicated in cases of paralytic or spastic talipes varus, and in some cases of congenital club foot. It should be performed only when a marked degree of correction is required, for the action of the muscle is so powerful as easily to produce an over-correction. When slighter grades of varus are present, the extensor proprius hallucis should be used instead of the tibialis anticus. The two operations are so nearly alike that the description of the one suffices.

"1. A 4 cm. curved incision is made over the insertion of the peroneus tertius. Skin, fascia and subcutaneous tissue are retracted to form a flap.

"2. The tendon of the peroneus tertius, which in the dissection of 25 cadavers and in all the operations was invariably present, is then slit for several centimeters and the metatarsal bones grooved for the reception of the tibialis anticus.

"3. An incision is made in the course of the tibialis tendon from the upper pole of its sheath, 3 to 5 cm. above the malleolus, to its insertion. The sheath is opened near its upper pole. Here again the exact knowledge of the sheath, its limits and inner architecture is necessary for the neat execution of the operation.

"4. It will be remembered that in describing the first operation attention was called to the fascial relations of the three anterior muscles of the foot; the tendons above the upper pole of the tibialis sheath lie in the same fascial compartment, then for several centimeters the extensor proprus hallucis and the extensor longus digitorum lie in the same fascial compartment separated from the tibialis anticus by a fascial septum. From the level of the malleolus downward there are three such compartments, one for each of the three anterior muscles. The transfer of the tibialis tendon is best made above the fascial septum separating it from the extensors, since in this way danger of adhesion is entirely avoided.

"The lateral margin of the divided fascia just proximal to the upper pole of the tibialis sheath is retracted until the extensor longus digitorum is visible. This level lies above the upper pole of the extensor sheath. To enter the sheath the loose connective tissue surrounding the extensor tendons—the paratenon—is incised until the bare tendon cells are reached. A probe passed along the tendon is then certain to enter the sheath. Passing the probe through the sheath again calls for a knowledge of its inner architecture. All five tendons, the four extensors and the peroneus tertius are connected with one another by means of a common mesotenon. The operator must be careful to pass the probe superficial to the tendons; otherwise he will draw the tibialis anticus between the extensor tendons and thus tend to interfere with their function as well as its own. The probe is guided in the direction of the peroneus tertius, made to puncture the lower pole of the sheath, and to appear between the fascia and the tendon near its insertion.

"5. The sheath of the tibialis anticus is now open its entire length and the fascia below the sheath incised until the insertion of the tendon is visible. The tendon must be divided as near the bone as possible; otherwise, it would not be long enough to reach its new point of insertion. It is then threaded with stout chromic gut and freed from its mesotenon until it can run a direct course into the sheath of the extensor digitorum and peroneus tertius.

"6. The tendon is drawn through the sheath by means of an eye-probe and fastened to the bone and to the peroneus tertius tendon by the technique described in the first operation. Care must be taken that the course of the tendon is straight and that the muscle is not twisted."

"No. 13.—Four and half year old girl. This patient was brought for treatment with marked valgus deformity. The tendo Achillis group of muscles, peroneal muscles and extensor communis digitorum were fairly strong. The tibialis anticus, extensor longus hallucis and tibialis posticus were weak. A valgus brace was worn. The deformity had existed for three years.

"The peroneus tertius tendon was transplanted into the periosteum of the scaphoid bone. A silk ligament was passed from the tibia to the tarsal bones at the inner side of the foot. Seven years and one month after operation the foot is held in practically perfect weight-bearing position. Silk ligament is strong and functioning well. There is slight function in the transplanted tendon. No voluntary dorsal flexion but passive motion dorsally to a right angle is possible. No brace is needed. The patient's mother thinks the foot has been greatly improved and that there has been much improvement in form, stability and usefulness."

The above procedure is excellent for the condition described and for the age at which the patient came for operation. For a patient 10 years or more older, arthrodesis of the astragalo-scaphoid joint may be combined with tendon plastic procedures to great advantage. It is in these cases especially that prolonged plaster of paris fixation in the inverted position and subsequent brace wearing will contribute so much to the final result. Final treatment will usually consist in the wearing of the Thomas heel and the employment of exercises until adequate strength of the adductors and dorsal flexors can be demonstrated."

"No. 12.—Fifteen year old girl. This patient's condition before operation was as follows: Marked equinus position; great toe hyper-extended and flexed; extensor of great toe weakened; tendo Achillis of good strength; dorsal flexors of the foot weakened but not paralyzed. No apparatus had been worn. The duration of the paralysis was since babyhood.

"The main points of the operation were, extensor longus hallucis tendon transplanted into head of first metatarsal bone; arthrodesis of first phalangeal joint of great toe; tenotomy of tendo Achillis; plantar fasciotomy. Two years and nine months after the operation the foot is somewhat under-corrected for equinus deformity. Some calluses are still present under the heads of the metatarsal bones. Voluntary and passive motions in dorsal flexion are stopped apparently by a bony obstruction at 80°. Extensor longus hallucis tendon is functioning well in its transplanted position. Slight voluntary adduction and complete voluntary abduction of the foot are possible. No apparatus is worn. Patient says there has been an improvement in her walking after the operation."

For hyper-extension of the great toe (and other toes as well) the transference of the point of insertion of the extensor to the head of the metatarsal bones has proven to be one of the most satisfactory of the surgical procedures about the foot. Degrees of disability out of all proportion to the apparent importance of the deformity occur as a result of these conditions and are greatly relieved by this procedure. Combined with this, it is often desirable to perform a resection of the first joint and secure a straight, rigid toe from which the painful dorsal callus has been entirely removed.

There has been some difference of opinion over the measures to be employed in the musculo-spiral paralysis and paralysis in the post-interosseus distribution in the forearm and hand. Arthrodesis of the wrist with the hand in the dorsiflexed position has not been generally approved. In the writer's experience, however, several patients have been given hands that were somewhat useful in exchange for hands that were of no use whatever by this procedure. In several other cases hands in which there was marked contracture deformity in the wrist with the flexor carpi radialis and the flexor carpi ulnaris chiefly at fault transference of the tendons of these muscles to the dorsum of the wrist has given the power of active dorsiflexion of the hand. This procedure violates the dictum that flexors should not be called upon by tendon transference to function as extensors. In this particular instance, however, the procedure has been done by a number of good operators and the results seem to justify the violation of the rule.

Nerve transplantation has been tried by a number of operators and the reports have been extremely variable. A number of entirely reliable observers condemn the procedure without reservation and it must be said that depriving certain muscle groups of innervation in an effort to switch it to others where securing the function is at least doubtful is a questionable procedure. To us it seems that some of the favorable reports may after all be explained by the unexpected restoration to function of muscles mechanically relaxed and supported by the mechanical after-care naturally incident to such a procedure. On the whole it must be set down that nerve transplantation as a procedure for the relief of these cases is still on trial and that it is to be recommended rarely and tried only under quite exceptional circumstances.

Arthrodesis has been extensively used and has given many excellent results. Moreover, this is a procedure which has a field of usefulness for those cases otherwise most hopeless; that is, where there is complete paralysis around a joint (flail joint). More recently, arthrodesis has also been used with tendon transference as an accessory procedure and has given useful extremities which were practically hopeless otherwise. Arthrodesis has found its greatest field for usefulness in flail ankle in connection with which it will be described later, as will also the Whitman operation, a modified arthrodesis with astragalectomy. Arthrodesis has also been used occasionally in other joints and will also be discussed for the treatment of special regions.

In this procedure the joint is laid open and all the exposed articular surfaces carefully removed. With approximation of these surfaces there is either complete ankylosis or fibrous union, so that complete or partial fixation of the joint in the position desired is obtained. In this as in other procedures of the same nature, exacting surgical and aseptic precautions are necessary and must be observed not only to obtain results but to avoid complications which would disable the patient and endanger his welfare much more than the original condition.

After arthrodesis as after any operation affecting a joint immobilization of the most perfect kind is important. The ultimate result certainly depends upon the careful maintenance of the desired position. The danger of post-operative inflammation is materially lessened by maintaining the parts in perfect rest. And finally, the patient's suffering during the first few days is reduced to a minimum by the application of splints or plaster of paris casts which provide against motion irritation at the site of the operation.

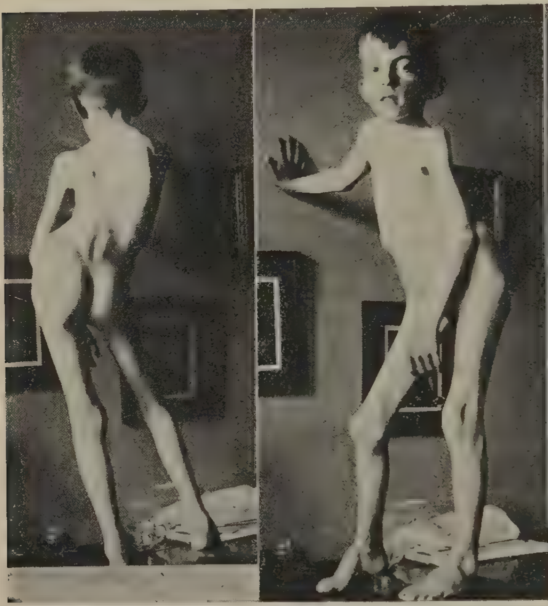

Fig. 328.

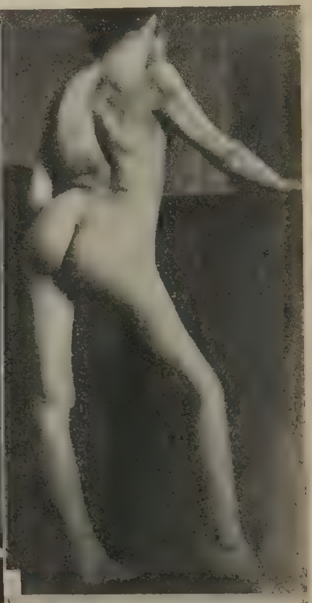

Fig. 329.

Figs. 328, 329.—Conditions like the above call for multiple surgical procedures and prolonged and careful post-operative care if the patient is to be greatly improved. Both the trunk and the right lower extremity in such a case as this may be materially benefited by the procedure devised by Dr. Soutter of Boston. In this operation the points of origin of the abductors and flexors of the thigh are moved downward upon the ilium until almost any degree of deformity like the above can be fully corrected. For paralytic deviations of the spine like the above which have become fixed it is now being advocated that open division of all muscular attachments on the convex side of the spine be made, the spine straightened to as great a degree as possible, and then arthrodesed by the Hibbs method or by a bone transplantation. This is a method which has not been employed by the present writer but which will doubtless be given serious consideration in the treatment of these cases which are so extremely difficult to treat after the deformity has once been permitted to develop. Figure 329 is submitted to show a patient in whom many elements of the deformity are similar to Fig. 328 but in which the spine deformity is exactly opposite.

"No. 19.—Twenty-nine year old woman. This patient had valgus deformity and poor stability of the foot. There was no power in the anterior tibial muscle or peroneal group. The tendo Achillis group was weak, also quadriceps weak in the thigh. A valgus foot brace was worn. She had suffered from paralysis for many years.

"The operative procedure was arthrodesis of the ankle joint and fracture of the lower end of the fibula. Five years and two months after the operation there is firm union in the ankle joint with a little voluntary and passive motion at the medio-tarsal joint. Stability and weight-bearing position are excellent. A little cavus deformity persists. Patient wears a plate. She thinks the operation a great benefit, and has not missed a day's work for four years. From an orthopedic standpoint the result is excellent."

"No. 25.—Sixteen year old girl. This patient presented moderate valgus deformity. All lower leg muscles were weak and there was moderate hyper-extension of the great toe. The hip was dislocated and quadriceps partly affected in the thigh. No brace was worn. The duration of paralysis manifested since babyhood.

"Astragalectomy was performed. Two years later, extensor longus hallucis tendon was transplanted to head of first metatarsal bone, and first phalangeal joint of great toe stiffened. Six years after her first operation the foot shows a good position but is rather unsteady in weight-bearing. There are a few degrees of passive motion at the ankle. The position of the toe has been improved, but some motion in the first phalangeal joint of the toe still exists. No braces are worn. Patient says she is worse than before operation because of the instability of the foot."

Cases 19 and 25 are presented together for comparison. The inference, which is borne out by other similar cases, is that arthrodesis in such cases is to be preferred to astragalectomy without it. Post-operative splinting for a considerable period (six months at least) is necessary for the best ultimate result. Any procedure which leaves the foot drop unrelieved or the ankle unstable from side to side is likely to prove disappointing.

"No. 18.—Seventeen year old boy. This patient applied for treatment with marked valgus deformity. Anterior part of the foot was abducted. Lower leg muscles were all weak; tibialis anticus and tibialis posticus muscles, weakest; peroneal group, strongest; tendo Achillis group and extensor communis digitorum, intermediate in strength. He walked with crutches and very little weight-bearing on affected foot. He had been paralyzed since babyhood.

"Arthrodesis of the ankle joint, and arthrodesis of astragalo-scaphoid joint were performed. Peroneus tertius tendon was transplanted into scaphoid bone. Seven years and three months after operation the patient reports by letter that his foot turns in as badly as before the operation. He used crutches for six years, then his local shoemaker made him a shoe which he uses with a brace and a cane. He returned twice to the out-patient department where he was fitted with a brace, after which he was lost sight of entirely six weeks after the operation. He says the operation did him no good, although he is no worse off apparently. Lack of supervision and post-operative care seem important factors in the late results of this case."

Three months of plaster of paris with the foot mildly inverted and three to six months longer of an outside iron with inside ankle strap would have been a minimum requirement for the post-operative care of this case. The operation was well selected and the result failed entirely on the point of failure in the post-operative care. The strong peroneal group rendered this case of especial importance as without such splinting valgus was certain to recur.

Arthrodesis of the elbow has been done repeatedly and has found favor with such an authority as Sir Robert Jones. Sir Robert Jones prefers, however, to fix the elbow at approximately a right angle by the removal of a diamond shaped skin area from the flexor surface of the elbow and by suturing the upper portion of the forearm to the lower portion of the upper arm in such a way as to maintain the arm in permanent flexion at about a right angle.

Osteotomy and **osteoclasis** are principally useful to correct gross deformities and to restore distorted parts to positions in which function is again possible. Contracture deformity must have become severe or distortion of the joint quite considerable in order to justify this procedure, but it is frequently indicated and often gives quite brilliant results. The account of the following case illustrates this.

F. J. was admitted to the hospital at the age of 12 having been paralyzed for five years. She could just get about on crutches, there being severe drop foot of both feet, the left extremity, on the whole, being more severely paralyzed. The left leg practically dangled from the hip—no weight whatever being borne upon it as she walked about on crutches. The right lower extremity, the foot and ankle being quite flail, the foot pronated badly when the patient walked. There was marked recurvation of the right knee and the knee was bent badly outward.

The surgical problems in this case were so numerous that she has naturally been treated in stages.

The attempt was first made to accomplish something by tendon transferences and fixations. The tendon transferences failed, however, largely because even the tendons transferred had too little power to render them useful in the new positions. Eventually she has had arthrodesis of both ankles, osteotomy of the femur in the lower third and arthrodesis of the left knee. She has improved to the point where she walks extremely well with crutches bearing the weight equally on both feet. She can walk without crutches using a cane, and does so to some extent. Her general health is excellent, bearing no ill effects from the rather long hospital treatment and repeated surgical procedures.

Post-operative splinting is of equal importance with adequate splinting immediately following the acute attack of infantile paralysis. In contracture deformities or serious deviations from the line of an extremity from whatever cause, the correction must be maintained and the extremity guarded by suitable apparatus promptly and correctly applied. Exactly the same appliances are useful here as in the first attempts at walking after the acute attack, especially after tendon transference, tendon suspensions, and arthrodesis. Adequate splint protection must be provided if the result of the operation is to be preserved. Plaster of paris, caliper splints, inside and outside irons, must all be employed as indicated long after the healing of the operative wounds has occurred. This is a point too often disregarded.

All of the treatment for these patients, at whatever stage, is reconstructive and almost entirely the responsibility of the surgeon. In the army reconstruction hospitals, however, there was a constant tendency to distribute the responsibility for the reconstruction of wounded soldiers among several different departments and department heads.

It is the principal message of this thesis to argue that the whole program of reconstruction with regard to infantile paralysis shall be largely or wholly worked out under the direction of the surgeon to whose care the patients are entrusted. This is important for several reasons. First of all, it must be the surgeon who sees the physical possibilities of the patient as he first presents himself for treatment. If the surgeon abandons the patient, or is deserted by him, at any stage short of realization of these possibilities, the patient has failed to gain as much as he might and the surgeon has failed to discharge his whole duty.

Sir Robert Jones has expressed this in a word by saying that what these patients must find in the surgeon to whom they come for relief is an "orthopedic conscience." From a very long observation of orthopedic patients and of what they have received and have failed to receive at the hands of hundreds of general practitioners and specialists, it is the conclusion of the writer that this orthopedic conscience is a thing far more important to strive for than any number of special methods of diagnosis and treatment.

The infantile paralysis patient who usually presents himself to the orthopedic clinic, dispensary, or hospital is one with much disability, contracture deformity, and delayed physical and mental development. His education is suffering for help from the standpoints of profession, hospital, and school. It has also been observed that even if these patients have been benefited by surgical treatment or hospital care, or by the application of splints and braces, they are several grades behind their fellows in the ordinary schools. They are therefore unwilling to return to the classes in which they would then find themselves. This is especially true if they have had hospital care for several months or spent a year or two in an institution where no educational opportunities were offered.

The writer is firmly convinced that the hospital which is well organized for instruction is the ideal institution for all of these patients. At the very beginning, such an institution attracts patients, because of the educational feature. It has been observed on many occasions that patients are much more easily persuaded to enter such a hospital and to remain for a fit period for treatment simply because it was possible for them to make up two or three or even more grades of school when they had fallen behind. It is perhaps unnecessary to remark that the hospital in which adequate instruction is provided is also a much easier hospital to operate because of the occupation of the patients and because the school atmosphere contributes to ease the discipline of the entire institution.

At the end of the period during which actual hospital care is required, it is still important, however, to provide what might be called the hospital school. In this institution the school atmosphere may predominate. Teaching may and should take the direction of preparing physically handicapped children for definite occupations, and the patient should not leave this institution until he is able, for his age and progress otherwise, to take rank with his normal fellows. For many of the patients, this means that they must remain until they have established themselves in a vocation at which they can earn a livelihood.

As suggested by other writers on the subject, this presupposes also what may be called an employment agency and social service department which will follow these patients until their ability for independence has been established.

It is not a secret that in spite of the elaborate organization and equipment provided for vocational training in the military hospitals much less was accomplished than had been hoped for in the way of returning our crippled soldiers to civilian life healed and independent. One might infer from the literature on the subject that England, France, Italy and the United States were prepared to send back to civilian life every man who was wounded—either healed for return to his previous occupation or specially trained for another vocation suited to the disability which he suffered during military service. This is very far from true. The point of failure, however, was not in the plan, organization or equipment of the hospitals or the hospital schools. They were admirably conceived and splendidly conducted. The point in which the program fell down was in the soldier himself.

Most of these men because of their age, lack of previous training, habits and disposition, failed to appreciate and to take advantage of the opportunities offered. In the British hospitals every expedient of persuasion and discipline succeeded in arousing the enthusiastic interest of a very small percentage only of the wounded soldiers in orthopedic hospitals. Too large a number were satisfied to return to their homes and accept the war office allowance and the allowances of their local pension committees rather than to exert themselves to take up their previous occupations or to train for new ones.

The same thing has been true to a certain extent of the soldier in this country. The answer to the difficulty lies in the proper management of our infantile paralysis and other civilian cripples during the years when they are susceptible to education and training.

In dealing with children instead of soldiers we find exactly the opposite to be true. The child who fails to be educated or to take advantage of opportunities for education and training is the exception and not the rule. Even these when young can have occupations drilled into them which later they could not possibly be made to acquire in any way.

In considering occupational or vocational therapy for children in hospitals or hospital schools it must, as in the military reconstruction hospitals, be considered from two standpoints: First, the purely physical for its therapeutic effect; and second, the vocational training which leads to future self-support of the individual. Both of these may be kept in mind.

There should be classroom and bedside instruction in the hospital. Occupations especially adapted to develop paralyzed or operated hands, arms, feet and legs may be employed at any stage, but even the early occupations may be selected with the end in view of training the individual for a special vocation.

A word may be said regarding the kind of institution best suited for this work. Splendid work has been done by small, privately endowed hospitals. A great deal of work has been done in special wards or departments of large hospitals. It has been demonstrated to the writer's mind, however, that the best results for these patients can be obtained only in the special hospitals with specially trained assistants and with adequate special equipment. It is the writer's belief that the most economical and most efficient hospital of this kind should be a 500 bed hospital, the chief surgeon and superintendent of which should be the same individual. Two competent orthopedic surgeons should be assistants and there should be the usual staff of head nurses, matrons, nurses, teachers and other assistants.

The department of physiotherapy should be complete and there should be a department of occupational therapy, including workshops, in which are taught all of the grades at least up to high school and such trades as carpentry, shoemaking, engraving, typewriting, bookkeeping, weaving, and brushmaking. In every group of patients selected for instruction in these shops will be found one or two of particular ability and of special promise. Moreover, by selecting these patients and by supervising them they will develop not only in independence, but physically as well.

The time of their operations, the selection of their treatment, the choice of occupations and the time of discharge from the institution should all be determined by the surgeon-superintendent, who alone by training and long experience is competent to decide these questions. It is not necessary that he himself should continue to perform surgical operations.

Our military experience demonstrated in all departments the value of the trained consultant. In civilian surgery wider range of opportunity both for determining the care of patients and for influencing to some extent the activities of physicians and surgeons not trained in special departments would certainly contribute to the welfare of many patients who are now receiving less than they might under the present plan.

For infantile paralysis and for other reconstruction the plea is for special hospitals large enough to devote the time, attention, and expenditure necessary, and in which specially trained officers and assistants may influence a sufficiently large number of patients to actually assist in solving one of our most serious present social problems; that is, the problem of the patient with infantile paralysis or otherwise crippled and who needs the best care that can be given by all that is included in true reconstructive surgery.

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CHAPTER CXXI

CEREBRAL SPASTIC PARALYSIS

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Cases of cerebral spastic paralysis due to hemorrhage occurring during or immediately after birth are recognized very infrequently by the attending obstetricians. A "blue baby," or one who does not cry promptly after birth, or who takes the breast with difficulty, should be subjected to a careful examination. In most cases of hemorrhage there will be some evidence of spasticity within a few days after birth, but in the vast majority of instances the signs are not noticed because they are not looked for with sufficient care. It is probable that a large number of persons who now drag out an unhappy existence with the handicap of spastic paralysis combined with more or less mental impairment could have been converted into normal individuals by an early diagnosis and prompt surgical intervention. The hemorrhages are almost always just above or just below the dura, and the clots can easily and safely be evacuated by operation, and the vessel, if still bleeding, can be tied.

Under conditions as they now exist, however, it is not to be expected that any radical changes will take place in obstetrical practice, and the orthopedic surgeon will continue to see almost as many cases of cerebral spastic paralysis as in the past. Most of them will be due to intracranial hemorrhage; some will be due to hydrocephalus or the other usual causes; many will have been acquired after birth.

The symptoms are very much alike, whatever may have been the etiology; they vary in degree and in location, but they all present a lack of coördinating ability in some or many muscles. With this is combined, in well-marked cases, deformities which occur from the unopposed pull of over-stimulated or under-inhibited muscular action. The hemiplegic cases are about three times as common as the paraplegic and diplegic (Peterson), and are more often acquired than congenital. They are less apt to show mental impairment than are the paraplegic or the diplegic. Their physical disability is less, and they can be improved to a greater extent by treatment.

Clinical Pathology.—The conditions found at examination depend upon the extent of the cerebral hemorrhage or the amount and location of the undeveloped or pathological tissues of the brain. For example: A small hemorrhage from the longitudinal sinus at the vertex of the skull will cause a spastic paralysis of the leg on the side opposite to the extravasated blood. The motor centers for the arms and legs lie along the fissure of Rolando in the precentral gyre, the thigh center being at the upper part, at the vertex of the hemisphere. The centers for the leg, foot, arm, forearm and hand follow downward and forward along the gyre. Still farther down are the centers for the muscles of the eyes, face, tongue and larynx, the latter center bordering upon the fissure of Sylvius. If, now, the hemorrhage at the vertex of the skull be sufficiently extensive to exert pressure upon the brain on both sides of the longitudinal (superior sagittal) sinus, the motor areas of both legs will be compressed or irritated, and a spastic paraplegia will result. A larger hemorrhage will extend farther down on both sides, and may involve the arm centers. If still larger, the eyes, face and tongue will be affected.

These hemorrhages are caused by the overlapping of one parietal bone upon the other during the passage of the child through the parturient canal, a small rent being torn in the wall of the sinus. Since the blood pressure in this large vein is very low, the hemorrhage does not usually cause death. The child is cyanotic at birth, and may require considerable stimulation before breathing becomes established. It is apt to be stuporous, and learns to take the breast with difficulty. Careful examination may reveal typical spasticity, but all too frequently the entire symptom-complex remains unrecognized.

The middle meningeal artery is torn in some cases, usually during difficult labors where the application of forceps has been found necessary. Arterial hemorrhage is more serious than venous, and more often causes death or the severer forms of paralysis. If of limited extent, a hemiplegia will result, although the initial paralysis may appear more general.

Spastic paralysis occurring in infancy or childhood may be due to embolism, thrombosis or hemorrhage in the course of such diseases as whooping-cough, scarlet fever, measles, and the like, and is not ordinarily amenable to cranial surgical interference.

Clinical Varieties.—In the ordinary types of hemiplegia, the history shows that the child was normal in the first few years of its life. Then occurred some form of disease, perhaps one of the exanthemata, or, more commonly, whooping-cough, during the course of which the child became unable to use the arm and leg with the former facility. More or less mental impairment may have coexisted, and the eye on the same side as the paralysis may have shown a strabismus. In some cases, recovery may have been nearly or quite complete, but the deep reflexes usually remain exaggerated. The arm is generally flexed at the elbow; the wrist and fingers are flexed, the thumb is adducted across the palm, and the wrist is in complete pronation. When the attention of the child is diverted, the contracted muscles relax and the flexion and pronation deformities disappear, but on requesting the child to perform any act with the hand, an immediate spasticity appears and grows greater in intensity in direct proportion to the voluntary effort which is attempted. If the examiner takes the child's hand and makes steady

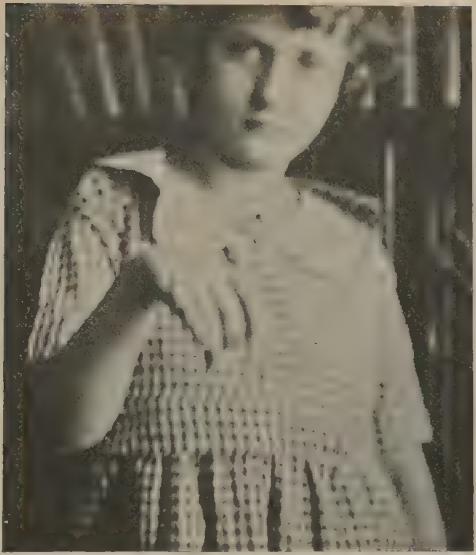

FIG. 330.—Spastic monoplegia.

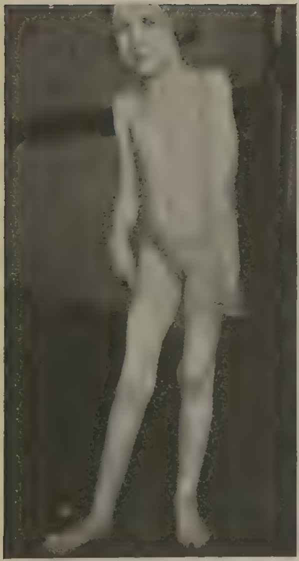

FIG. 331.—Spastic hemiplegia.

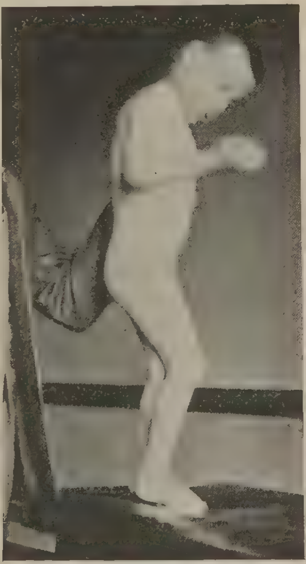

FIG. 332.—Spastic diplegia.

and firm pressure or traction, the spastic muscles will slowly yield without pain or discomfort, and the over-correction will easily be possible. If, however, no treatment be used, the muscles may become firmly contracted in the course of a few years, causing a permanent deformity which can only be relieved by surgery.

The lower extremity in the hemiplegic cases shows equally characteristic malpositions. The knee is flexed, and often cannot be voluntarily extended. The calf muscles, chiefly the gastrocnemius and soleus, are contracted, and pull the heel upward, causing the deformity known as equinus. In most cases the foot also assumes either a varus or a valgus position, and may readily become severely deformed. The thigh may be adducted by the pull of the three large adductors, but this deformity is not so common in the hemiplegic cases as in the paraplegic. When the child attempts to walk, the gait is clumsy and spastic. The knee is held flexed, and the child walks on the ball of the foot, with the heel not touching the floor. Unless these contractions be overcome by operation or treatment, they will, after a time, become permanent contractures.

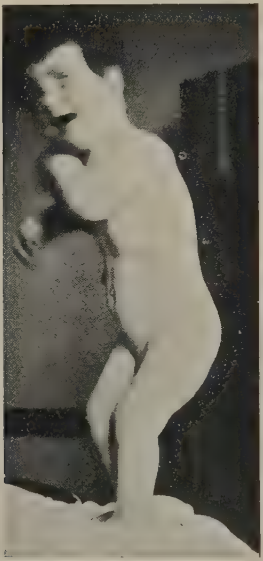

FIG. 333.—Spastic diplegia.

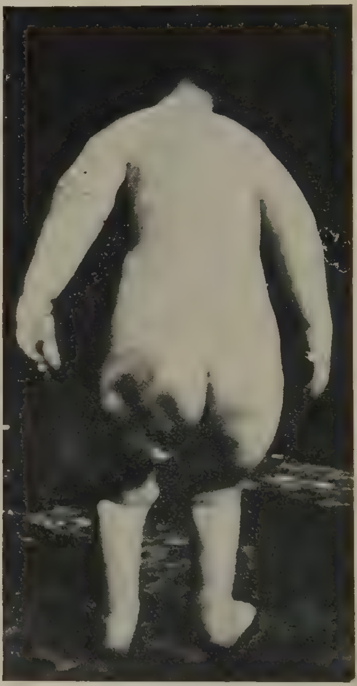

FIG. 334.—Spastic diplegia.

The cases of paraplegia, commonly called Little's disease, are always slow in learning to sit up and to walk. Many of them are brought to the physician for examination simply for this reason, no other abnormalities having been noticed by the parents. The child may be of such low-grade mentality that it is practically or actually an idiot or imbecile, but the parents usually do not recognize this fact, and often assert that it is unusually bright and clever. This attitude of the parents is probably in the nature of a "defense reaction." On the other hand, many paraplegic children have little or no mental deficiency. The knee jerks are exaggerated. There is usually no ankle clonus, and this point is of some value in differentiating the condition from a spastic paraplegia due to compression of the cord from the effects of tuberculous spondylitis. The thighs are flexed and adducted, sometimes crossing one over the other (scissor-legged); the knees are flexed, and the feet are drawn down by the contraction of the calf muscles. When walking is attempted, it is spastic in the extreme. The knees knock together, or cross over each other, and are flexed. The feet assume the equinus position, and the weight is borne on the metatarsal bones, with the heels not touching the floor.

The diplegic cases combine the peculiarities of the paraplegic and the upper extremity symptoms described under hemiplegia.

Treatment.—In discussing the treatment, the mental condition of the patient is of the first importance. It is of little use to attempt by orthopedic measures to improve the locomotive powers of a hopeless idiot or imbecile. From a eugenic viewpoint it is probably better that such an individual remain unable to mingle with the rest of the world. If there is no possibility of restoring a reasonable degree of mentality by a cranial operation or by medication, this patient should remain untreated.

Cases in which the intelligence is relatively normal can be greatly benefited by properly conducted treatment, but it must be explained to the parents or guardians that, no matter how successful the treatment, there will probably never be a complete recovery from the spasticity. The child may become able to walk with great ease, and for long distances, but will usually not be a good or graceful runner, except in rare instances. A spastic hand may become useful in ordinary work, but is rarely capable of the finer grades of coördination. In practice, it is wise to give this information with tact and discrimination, so as to avoid shocking too severely the anxious and despondent parents. This can usually be done by explaining that the child has suffered a serious injury to the brain, and that too much must not be expected or required at first; that for a long time the child will not be able to progress as rapidly as a normal individual, and that every allowance must be made for the apparent backwardness of development. Unless this, or a similar, approach be used, the parents may become so discouraged that they may fail to coöperate in the long and tedious treatment, or, on the other hand, they may believe that the child has only a mild and transitory defect which really needs little attention.

During infancy, attempts may be made to stretch and manipulate the arm and leg, and splints may be applied to maintain an improved position, but as a rule these attempts are not conspicuously successful.

In older children it is of considerable advantage to fasten the opposite hand behind the back so that the child will be forced to use the paralyzed one to the fullest possible extent. This excellent method is very commonly neglected in practice. but should always be followed. The results are sometimes amazingly good. Manipulations should be carried out several times a day, the hand being held fully supinated, the fingers and thumb extended, and the elbow straightened, this position being maintained for two or three minutes at a time. Similar exercises should be performed upon the leg and foot. If there is a marked tendency toward flexion of the knee and equinus deformity of the foot, a simple brace should be used to control the malpositions until the time has come for more radical treatment. Braces are never curative, but may prevent the formation of actual contractures of the muscles.

Operative treatment should not be undertaken, in ordinary cases, before the child has reached the age of five years and has learned to control the evacuation of the bowels and bladder. The simplest and safest form of operation is the division and lengthening of the contracted or overacting muscles and tendons.

Upper Extremity.—In the forearm, the pronator radii teres, the pronator quadratus, the palmaris longus, the flexor carpi radialis and ulnaris, and the flexors of the fingers usually need to be lengthened. An incision is made in the middle of the forearm, at the inner border of the radius. The deep fascia is opened, and the brachio-radialis muscle (supinator longus) is identified and retracted. Beneath it will be seen the flat and shining tendon of the pronator teres, running obliquely downward and outward around the shaft of the radius. The tendon is cut along the bone, and care is taken to be certain that all the fibers are divided. The incision can then be closed and sutured. Another incision is now made over the middle of the flexor surface of the forearm, just proximal to the wrist joint, about 3 in. in length. The palmaris longus tendon is cut straight across and allowed to retract. The tendons of the flexor carpi ulnaris and radialis are exposed and lengthened by the step method and sutured with fine chromicized catgut.

Whether or not the flexor tendons of the fingers and thumb should be lengthened depends upon the actual extent of the disability. There are nine of these tendons, and they are of very small size in children. It is no easy task to split them and suture them, and in many cases they become attached to each other by a mass of dense adhesions so that the individual movements of the fingers are greatly impaired. It is probably advisable to defer this portion of the operation until the patient has reached the age of 12 or 14 years, except in extreme cases.

A. H. Tubby advises an attempt to convert the pronator radii teres into a supinator, by detaching it from the radius and passing it around the radius on the opposite side, reinserting it into the bone by a suture through a drill-hole. This operation has been performed many times in America, but the results have not seemed to be noticeably better than when the tendon is simply divided.

The pronator quadratus is not often divided by American operators, but in severe cases it should be exposed through the original incision used for the division of the flexors of the wrist. It is a broad, flat muscle, and can easily be identified and severed. In some cases, the flexor carpi radialis and ulnaris have been converted into accessory extensors by carrying them around to the back of the wrist and suturing them into the tendons of the extensor carpi ulnaris and radialis longior.

Lower Extremity.—The tendo Achillis requires division in nearly every case. This tendon should never be cut by a simple transverse tenotomy, but should always be exposed by an open incision and split down the middle, either sagittally or frontally, for a distance of at least 2 in., and the ends sutured together in an overlapping manner with fine chromicized catgut. Care must be observed to avoid too great lengthening, 1 in. being sufficient in the ordinary cases. Otherwise, if the tendon be too much relaxed, the opposite deformity may develop, with the foot pulled up into dorsiflexion, or calcaneus position.

The hamstring tendons should almost always be divided, even though very little actual spasticity may seem to be present in these particular muscles. It often happens that, through over-cautiousness, or because there seems to be little actual indication, the tendo Achillis alone is lengthened, but, with few exceptions, it is later found that the hamstrings have become definitely spastic and require division. It is a safe rule to follow, that where the Achillis tendons need to be lengthened, the hamstrings must always be included in the operation. An excellent exposure of these structures can be obtained by a transverse incision in the popliteal space, at the level of the upper portion of the condyles. The biceps tendon must be exposed with care, and picked up on a blunt dissector, to avoid injury to the external popliteal nerve which lies directly internal to the tendon. The semimembranosus and semi-tendinosus are similarly exposed at the medial side of the popliteal space, and are divided transversely. The gracilis is also cut. Some operators prefer to excise a section 2 or 3 in. long from all of these tendons. In very severe cases an actual tendon transplantation or transposition may be done, the biceps and either the semi-tendinosus or the semi-membranosus being dissected freely from the insertion to a point 5 or 6 in. upward in the thigh, cut off at the insertion, and passed forward through a channel made in the subcutaneous fat, one on either side of the knee, and inserted subperiosteally into the patella. This operation, which is of marked benefit in suitable cases of infantile paralysis (anterior poliomyelitis), is not of striking value in the spastic cases, and is not generally to be advised. The adductor muscles usually require division, and this may be done sufficiently thoroughly by subcutaneous tenotomy, the tenotome being plunged under the skin just below the symphysis pubis at either side of the median line. The legs are abducted to make the muscles tense, and they are cut through with a short, sawing motion, little by little. A thumb or finger is placed over the femoral artery to act as a guide. Unless the operator has had considerable experience, his courage is likely to fail before he has cut enough of the muscles. It is probably safer and wiser, at least in older children, to make an incision over the adductor group and bring the muscles up on a director into view and cut them under the guidance of the eye.

After the operations are finished, a double plaster of paris spica cast is applied, with the legs widely abducted and straightened at the knees, and with the feet at right angles to the leg. The cast will be much strengthened if a piece of lath or board be placed just above or below the knees, running from one leg across to the other, and fastened to the leg casts by many turns and moldings of the plaster bandage. This also makes a very useful handle for the nurse in caring for the patient. Windows are cut in the popliteal spaces and beneath the heels, so that the progress of healing may be watched. The cast is not removed for at least six weeks in the milder cases, to three months in the most severe. Massage, manipulation, and especially active muscle training, are then begun, and continued for as long a time as may be necessary. The child will have to be taught to walk, and this may take many months of patient endeavor. Children differ very materially in their ability to learn to walk, and in some cases the results, for a long time, may be disappointingly slow, but sooner or later nearly every individual will become able to get about without assistance. This statement, of course, is contingent upon the relative mental capacity of the patients, and the care exercised in selecting suitable cases for operation.

The operative treatment above described is regarded as being the safest and sanest method at present available, and it is considered that the results, as a whole, are at least as good as those obtained by any other method or technique. A brief description, however, will be given of the other forms of operation which are at present being employed in various clinics and centers. Foerster's operation: Foerster, of Breslau, many years ago advocated the exposure of the spinal cord by laminectomy, and the division of the posterior (sensory) nerve roots from the second lumbar to the second sacral roots, inclusive, the object being to inhibit muscular contractions by removing the peripheral sensory stimuli. The operation is much more dangerous than tenotomy and myotomy, and the results do not seem to be sufficiently favorable to warrant its employment.

Alcohol Injection of Nerves.—Allison and Schwab advocated the injection of the peripheral nerve trunks (obturator, hamstring supply, and posterior tibial, for example) with 85 per cent. alcohol, thus producing a complete paralysis of the muscles supplied by these nerves. This paralysis lasts from 6 to 12 months, during which time the antagonistic muscles are to be trained and exercised so that they can successfully function after the return of power to their adversaries. The operation was performed by many surgeons, but is not now generally used.

Stoffel's Operation—Partial Neurectomy.—The nerve trunks are exposed (median, sciatic, posterior tibial, obturator, for example), and the various fibers supplying the over-stimulated muscles are carefully traced down. A portion of the nerve supply of each muscle is then exsected, thus weakening to any desired degree the actual power of the muscle. An electrode is used to determine accurately the distribution of each nerve bundle, although a very fair idea of the distribution may be gained by gently pinching the nerve with a pair of fine, smooth-pointed forceps. A thorough knowledge of the anatomy, and a rather wide dissection, are necessary. Several American surgeons, notably A. Bruce Gill, of Philadelphia, have written favorably of the results, but the operation has a basic and fundamental weakness in that it is a destructive operation, and its effects, if too extreme, cannot be repaired. It is also not sufficiently exact in its quantitative determination, since it is impossible to estimate definitely how much of the nerve supply should be sacrificed.

Intraperineural Neurotomy.—J. J. Nutt has advised direct transverse section of the nerves within their sheaths, with the object of producing a complete temporary paralysis. His first paper was followed some years later by another article, but no other surgeons have corroborated his conclusions. The idea is similar to the alcohol injections, and the method will probably have the same fate.

In conclusion, it is safe to say that most cases of cerebral spastic paralysis in children will improve with the passage of time; that all of them who have reasonable intelligence should be assisted by careful training and, if necessary, by well-planned and well-executed operations, and that, of all the operative methods so far proposed, simple tenotomies, myotomies, and muscle and tendon lengthenings are the safest, sanest and best.

CHAPTER CXXII

TUBERCULOSIS

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Evanston, Ill.

Introductory.—Although our knowledge of tuberculosis has made progress during the four decades that have passed since Robert Koch discovered the bacillus of tuberculosis, the white plague still claims more victims than does any other disease.

During these years we have learned:

1. That infection with this bacillus is much more prevalent, especially among children, than was once believed.
2. That not only the human, but also the bovine type of bacillus is pathogenic for man.
3. That the aspiration of bacilli, disseminated by tuberculosis patients is the most frequent mode of infection.
4. That the primary focus is usually found in the lung.
5. That rest, light, air and a carbohydrate-low diet are our chief therapeutic measures.

This disease is still rampant, in spite of the above known facts, and a multitude of other discoveries, because:

1. Infants and young children are very susceptible.
2. Early diagnosis is difficult.
3. The sources of infection are manifold.
4. The course of the disease is often chronic.
5. Specific prophylaxis and therapy are still unsuccessful.

The National Tuberculosis Association places the annual death rate from all forms of tuberculosis in the United States at a hundred and fifty thousand, the diagnosed active cases at a million, and the unsuspected cases at two million.

The prevention, diagnosis, and cure of tuberculosis still are, therefore, the largest problems of medicine, and much remains to be done.

GENERAL ETIOLOGY AND THE TUBERCLE

General Etiology

Bacillus Tuberculosis.—The isolation of the etiological factor by Koch[47] was of immense theoretical as well as of practical value, for it finally proved that tuberculosis was not a constitutional, but an infectious disease. By special staining methods he found this microörganism in a great variety of diseased organs and tissues, grew it in pure culture and successfully inoculated susceptible animals from which the bacillus was subsequently recovered.

Morphology.—While the form, size and structure of the human type of bacillus may vary somewhat under different conditions, when obtained from sputum, spinal fluid and tissues, it usually appears under the oil immersion lens as a slender rod, slightly bent or curved, with a length of 2 to 4 μ and a breadth of 0.5 μ (Fig. 335).

Types.—Theobold Smith[79] was the first to prove that the bacilli found in tuberculous cattle and those found in patients with phthisis are not identical. He also pointed out that the bovine type of infection in man is usually of alimentary origin and localization, and mostly restricted to childhood. The bovine type of bacillus is usually shorter, straighter and thicker than the human. It is cultivated less readily and is much more virulent for cattle and rabbits.

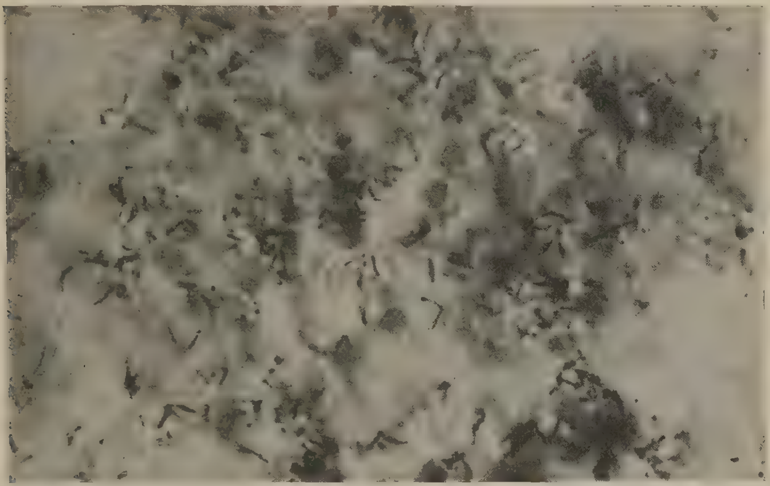


Fig. 335.—Bacillus tuberculosis (Typus humanus). Microscopic section of a tuberculous lung of a three months old infant. (Courtesy of Dr. R. A. Austin.)

Staining.—The examination of sputum or aspirated stomach contents is best carried out by a preliminary treatment of the material with antiformin, whereby most of the organic matter, including other bacteria is destroyed.

Technique.—Flood the fixed cover-slip or slide with Ziehl's carbolfuchsin solution and allow to gently simmer for about a minute; decolorize with acid alcohol (2 per cent. HCl in 80 per cent. alcohol), then wash and counterstain with methylene blue, wash again and dry.

Power of Resistance.—Although the tubercle bacillus multiplies only within a narrow range of conditions, its viability outside the host is relatively great. Moist sputum in a dark place may harbor virulent bacilli for months. Completely dried sputum, on the other hand, is rarely infective for more than 10 days, and direct sunlight kills the bacilli in dust within a few days. Gastric juice does not destroy all bacilli introduced into the stomach and virulent organisms may therefore pass to the intestine. The bacilli can withstand a dry temperature of 100°C. for an hour, but moist heat kills at 60°C. (140°F.) in 15 to 20 minutes. Pasteurization should, therefore, be carried out in closed vessels under such conditions. Boiling for five minutes also renders the germs inert. Butter made from unpasteurized cream from tuberculous cows contained virulent bacilli for over five months. Extreme cold is not germicidal. Five per cent. phenol added to sputum fails to kill in 24 hours, whereas the same strength of lysol is effective.

Predisposing Factors.—"Predisposed to tuberculosis" is a broad, often erroneously used term, which for many years handicapped our true understanding of the disease. Every human being is, in a sense, predisposed to tuberculosis, for the human race is very susceptible to infection with the human type of bacillus. We have specific biologic proof (diagnostic tuberculin test) that nearly all infants are born free from tuberculosis; also, the pathologic evidence that nearly every tuberculous infection is acquired from without some time after birth.

Geographical.—In a few parts of the world tuberculosis is still absent; namely, Iceland, Shetland Islands, parts of Norwegian coast, Transvaal, Zambesiland and South Sudan. In others, more recently infected, the death-rate is on the increase: Ireland, Tyrol, Japan. In France the death-rate is stationary, in England and the United States it shows a rapid decline. The tenement children of Vienna, Paris and London show a higher morbidity than do those of New York, Chicago, and St. Louis.

Hereditary.—In attempting to study the so-called inheritance of tuberculosis, one must realize that the re-appearance of the disease in successive generations or in families does not necessarily imply inheritance. Inherited, in the strict sense, is only that which develops from the determinants of the fused sex cells, ovum and spermatozoan, that which is imparted to the offspring through the germinal substance. We may speak of the "non-inheritance of a constitutional resistance" or of the "inheritance of scant powers of immunity" but not of the "inheritance of a predisposition." Prenatal infection is not inheritance. All that an infant possesses at birth is not necessarily inherited. Influences, such as the tubercle bacillus, operating on the fetus during the gestation period, are environmental, not hereditary. A debilitated mother may give birth to a frail infant with poor resistance, but dissemination of bacilli from an active tuberculous focus in the parent is the usual source of prenatal and post-natal infections.

Racial.—The Irish, Negroes, Indians and Italians in our country show a much higher incidence of the disease than do the Jews. In the latter this is interpreted as "racial immunity"—the result of centuries of civilization and urban life, whereby those with the greatest power to develop immunity are supposed to have survived and propagated. The problem is a complex one, involving previous and present modes of life, climate, change in occupation, and other factors.

Sociological.—An "acquired predisposition" may be the result of a decrease or absence of power to develop immunity, dependent upon a lowered resistance of the body forces. The children of the poor, especially those of tuberculous parents, show a high incidence of morbidity and mortality. Children living in congested cities, in industrial centers, in dark, close alleys, in basements and tenements where light, ventilation, and sanitation are inadequate, are very prone to the disease. Food and shelter are often deficient and intercurrent diseases such as rickets, pertussis, etc., are at work. In such an environment with its congested living quarters, one not infrequently finds also neglected, indifferent tuberculous adults. Pollak[69] found 97 per cent. of the infants of tuberculous parent infected by the end of the first year. The environment of the city poor is an important predisposing factor, the improvement of which would greatly reduce morbidity and mortality.

Age.—No age is exempt. Although clinical tuberculosis in infants under three months is rare, one may say that the younger the child at the time of infection, the more likely will the disease become active. Also, the older the child at the time of infection, the more likely will the lesion heal or become latent.

The many published statistics show considerable variation in regard to the percentage of infection and morbidity at the various periods of childhood. This variation is mainly due to: The locality in which the observation was made, the class of children included, and the methods used to make a diagnosis (cutaneous or intracutaneous diagnostic tuberculin test; macroscopic pathology or guinea-pig inoculation of suspicious lesions).

TABLE 1.—PERCENTAGE OF POSITIVE TUBERCULIN REACTIONS GROUPED IN AGE PERIODS

Age in years	Veeder and Johnston[80] (cutaneous and intracutaneous), St. Louis	v. Pirquet[67] (cutaneous), Vienna	Hamburger[35] (subcutaneous), Vienna	McNeill[53] (cutaneous), Edinburgh
under 1	1.5	0	0	14.1
1– 2	5.5	0	0	29.9
2– 4	19.0	13	26	46.6
4– 7	25.0	30	54	28.8
7–10	30.0	48	75	51.9
10–14	36.0	70	94	55.0

The above table incidentally illustrates the influence of locality and the test employed.

The following comparative table of age distribution also illustrates the vast difference which exists when only one cutaneous test or when repeated tests are resorted to:

Table 2.—Age Distribution of Patients Reacting Positively

Feer,*[24] per cent. positive	Age	Bass,†[5] per cent. positive
1.2	0– 6 months	7.8
6.0	6–12 months	15.8
15.0	1– 2 years	17.2
	2– 3 years	
20.0	3– 5 years	42.8
	5– 6 years	78.0

* Based on one cutaneous test on 4000 Zurich dispensary patients.

† Based on repeated cutaneous and intracutaneous tests, performed on 206 children in New York Home for Hebrew Infants.

The frequency of tuberculous infection as diagnosed by the tuberculin test shows a steady increase as age advances, but a true conception of incidence can only be formed when the test is repeated or when the more delicate diagnostic tuberculin tests are used.

Comby[15] found that of 1675 Parisian children, 638 (38 per cent.) showed tuberculous lesions at necropsy. The cases were distributed as follows: Under three months, 1.82 per cent.; three to six months, 18.18 per cent.; six to twelve months, 26.25 per cent.; one to two years, 46.72 per cent.; two to five years, 60 per cent.; five to ten years, 67.15 per cent.; ten to fifteen years, 71.23 per cent.

These figures do not represent the incidence of tuberculosis in the community, but the frequency in hospital patients. They probably show that the amount of infection among children varies in different localities. None of the above figures imply that death was due to the disease. All figures fail to inform us on the frequency of tuberculous infection of all children of a community at the different age periods.

Intercurrent Disease.—The child who has withstood the primary infection has elaborated sufficient immune-bodies to keep the process from becoming active. If this resistance against the bacillus is maintained at all times, he will not develop the disease. The relative frequency of active tuberculosis as a sequel to measles, whooping cough, influenza, etc., and the disappearance of the tuberculin reaction during and for some time after such infections leads one to conclude that intercurrent disease may lower the specific body-resistance sufficiently for a latent tuberculous infection to become active. Diseased tonsils and adenoids, recurrent bronchitis and frequent head colds may also possibly bring about a decrease in immunity.

Modes of Infection.—Hereditary Transmission.—In the true sense of the term, this does not exist.

Prenatal Infection.—Years ago, v. Baumgarten[6] set up an hypothesis that nearly all tuberculosis, even that recognized after many years, is due to the presence of the germ in the body before birth. The bacilli are supposed to accompany the sperm or ovum or to pass from the placenta to the fetus. The "inherited" germ may lie dormant in latent foci, possibly throughout the life of the host, or, as opportunity arises, may multiply and produce the disease. The low incidence of tuberculosis in infants who are at once isolated from their actively tuberculous mothers, the prevalence of negative tuberculin reactions in the newborn and the gradual increase in incidence with increasing age are proofs that congenital transmission of the bacilli is extremely rare.

Congenital infection, although rare, has been established by authentic case reports. The mother in most instances has suffered from the disease in an advanced form, and the infant is either stillborn or survives only a short time (see "Infantile Tuberculosis").

Germinative.—Even though intra-ovarian infection is conceivable, every possibility of such an ovum being fertilized is absent. The possibility of a germinative transmission through a bacillus-containing spermatozoan is also conceivable, but it has not been observed in man. Without tuberculosis in the mother, congenital tuberculosis is probably impossible. Many tuberculous women are sterile, or spontaneous abortions frequently follow conception.

Placental.—The normal placenta is a filter, impervious to bacteria. For infection to occur through it, a lesion must exist whereby a communication between maternal and fetal blood becomes established. The bacilli may be contained in the maternal blood or may exist in the diseased placenta. This mode of infection has received careful study by Schmorl and Kockel[77] and the literature is well reviewed by Weber.[81] There is no doubt that bacilli can be transmitted from the mother to the fetus if the barrier is broken. Tubercle bacilli have been found in the organs of fetuses and infants born of tuberculous mothers. Placental tuberculosis may occur, even in relatively mild cases of tuberculosis. The bacillus has been found in the placental blood spaces of phthisis and miliary tuberculosis cases without noticeable tissue changes. Any of these conditions suffice for the transmission of the bacillus to the fetus. Incubation period and primary focus are absent.

Intrapartum.—Transmission of the bacilli to the child may occur during birth if the villous vessels are torn on separation of the placenta, provided bacilli are present in the maternal blood or in lesions on the raw placental surface. Cases have been recorded in which bacilli were found in the blood of the umbilical cord and in the liver of the newborn of tuberculous mothers, without any other organic changes in the infant. Aspiration of bacillary amniotic fluid may also become the source of tuberculous infection during birth.

Post-natal Infection.—Most infants, including those born of tuberculous parents, are born free from tuberculosis. Exposure usually implies infection, and infection in the infant often signifies active disease. Early post-natal infection can produce marked pathology in four to eight weeks, for active and progressive lesions are characteristic of tuberculosis of infancy (see "Infantile Tuberculosis").

Inhalation.—Von Pirquet[68] estimates that probably about 95 per cent. of all tuberculous infection occurs by this route. It is variously known as the bronchogenous, aerogenous, or respiratory mode of infection. Cornet[16] laid stress on the inhalation of bacillus-laden dust as the mode of infection. He was able to produce the disease in susceptible animals inoculated with carpet-dust from rooms frequented by tuberculous patients.

Flügge,[27] on the other hand, thinks that the danger of infection from bacillus-containing dust is negligible. According to his *droplet theory,* infection is chiefly conveyed by the finely divided particles of bacillus-laden sputum disseminated by the cough of phthisis patients. It has been shown that the expired air of consumptives is not infective. The infectious droplets may float in the air for about 30 minutes, and are found only rarely at a greater distance than 3 to 4 ft. from the coughing patient. The danger of infection from this source is, therefore, circumscribed both in space and time. If the advanced state of the pulmonary lesion can be relied on as being the primary focus, then the careful studies of Ghon[29] and others show that most cases are of inhalation origin.

Ingestion.—Von Behring[7], Calmette,[12] Osler,[60] Cobb[14] and others give the intestinal canal first place as the portal of entry. It was pointed out that the number of bacilli swallowed is out of proportion to the very doubtful number that might reach the lung by inhalation. Bacilli may enter by way of the tonsils, pharynx, esophagus, stomach or intestine.

In 1903 von Behring[8] said that pulmonary tuberculosis could be induced through intestinal infection, and that infected milk fed to infants was the chief cause of phthisis in later life. Calmette[12] showed how the lungs may become infected through the intestinal route without leaving a trace of the disease at the portal of entry. Osler and McCrae[60] state "the bacillus prefers the throat and intestines in childhood, the bronchi and lungs in adults." Cobb remarks that all portions of the lungs are invaded by dust and tubercle bacilli in equal measure, whether the route is through the trachea or through the intestinal tract.

Von Pirquet,[68] Hamburger,[35] H. Koch[43] and Finkelstein[25] give the alimentary canal second place as a portal of entry for the tubercle bacillus. Alimentary tuberculosis is more common after the first year, possibly because more unboiled milk is given to children after that age. Intestinal, mesenteric and peritoneal lesions may also result secondarily from swallowed tuberculous mucus from the lungs. Animal experiments have conclusively shown that a relatively small number of bacilli are necessary to produce the disease by aspiration, while more than a million times that number ingested may fail to produce the disease.

The tonsils are occasionally the site of entrance. H. Albrecht,[3] Ghon,[29] and Chancellor[13] each describe such a case. The primary lesion shows ulceration and the regional lymph glands are intensely swollen. With the buccal mucous membrane intact, it is not likely that the tubercle bacillus can gain entrance.

Cutaneous Inoculation.—As the intact skin is impervious to the bacillus, trauma is necessary for an infection to become established. The small number of primary dermatogenous lesions reported in the literature is ample proof of the infrequency of this portal of entry.

Sources of Infection.—The tubercle bacillus is not ubiquitous in spite of the prevalence of active tuberculosis in man and domestic animals.

Man.—The main source of infection is the individual with active pulmonary tuberculosis, who, during long periods of the disease, expectorates enormous quantities of bacilli. Nuttall[58] estimates that a phthisis patient expectorates from 1½ to 4⅓ billions of bacilli each 24 hours. Unless rigid precautions are painstakingly enforced, these individuals become the main source of infection. Lamson[50] found that 67 per cent. of the individuals in families in which there had been a case of active tuberculosis gave evidence of infection; in families where there was a latent case, 22 per cent. had become infected, while in families free from the disease only 2.5 per cent. showed infection. Statistics show that infants infected by tuberculous mothers die earlier than those of tuberculous fathers, probably due to earlier more and massive infection, the result of more intimate and repeated contact.

The following observation by Reich[72] shows the danger of direct contact infection: A tuberculous midwife in a small town made it a practice to remove mucus from the mouth of the newly born by direct mouth-to-mouth aspiration. During 13 months 10 of these infants died of tuberculous meningitis. In none of these families was there a history of tuberculosis. In the practice of other midwives in the town, not a single child contracted the disease.

Contaminated Objects.—Cornet[16] said: "The consumptive himself is almost harmless, and only becomes harmful through bad habits." The children of the poor are so frequently infected and show signs of the disease because their mode of life so often leads to massive infection. Besides the patient himself, contaminated linen, walls, floors, carpets, dishes, spoons, drinking cups, etc., may be potential sources of infection. Children of the creeping age usually put fingers and objects into their mouths more or less habitually. Painstaking experiments by

Flügge[27] lead to the conclusion, however, that the occasional ingestion of small quantities of bacilli is probably not sufficient to produce the disease.

Stool, urine, perspiration, and pus from bone and gland discharges of tuberculous patients may contain virulent bacilli and can become as much a menace as bacillary sputum.

Household pets (dogs, cats and parrots) are subject to tuberculosis with the human type of bacillus and must, therefore, be considered added sources of infection.

Contaminated Food.—While a number of investigators have found no bacilli in the breast milk of tuberculous mothers, Kuraschiga, Mayeyama and Yamada[48] found tubercle bacilli in the breast milk in 10 of 13 incipient cases of tuberculosis and in all of the advanced cases they examined. It has been shown that occasionally cow's milk offered for sale in cities has contained tubercle bacilli of the bovine type in spite of commercial pasteurization. For this reason, all milk should be again pasteurized or boiled before it is fed to infants and young children. Cream, butter, ice-cream and cheese may also harbor virulent bacilli of the bovine type unless proper precautionary measures are resorted to. The investigations of Park and Krumwiede[61] in New York show that tuberculosis of the lungs, meninges and osseous system in children is nearly always of human origin, but if in the intestines or cervical glands, it is not infrequently due to the bovine type. In children under 5 years, in 76 of 367 cases, and in children from 5 to 16 years, in 45 of 176 cases of tuberculosis, the bovine type was found to be the etiological factor. Frazer,[28] studying bone and joint tuberculosis in Edinburgh, found that in 62 per cent. was the disease due to the bovine type. Other English observers, including Griffith,[30] find the bovine type of bacillus a rather frequent etiological factor. Probably more raw milk is consumed there than in New York. The meat of tuberculous animals is rarely dangerous if well cooked.

The Tubercle.—The primary localization of the tubercle bacillus is usually in lymphatic structures, where, about the focus of infection, the bacilli multiply and epithelioid cells and polynuclear leukocytes appear in great numbers. Many of the latter undergo rapid destruction and as the tubercle grows, lymphocytes increase, predominate and persist. Giant cells, huge, multinuclear masses of protoplasm, then usually make their appearance. The tubercle is, therefore, a circumscribed inflammatory growth varying in structure and size according to its age. The nodule, in the course of its development, undergoes a characteristic degeneration and coagulation necrosis in its center, produced by the bacilli or their products. The tubercle is thus gradually converted into a conglomerate yellowish-gray mass in which bacilli, but no blood-vessels, are to be found. These cheesy bodies may soften and ulcerate or heal with sclerosis and calcification. This caseous degeneration of the granulation cells is characteristic of all tubercles. The course is probably determined by the number and virulence of the bacilli and the amount of resistance of the body-cells.

The order in which the various organs are most frequently involved is: Lungs, bronchial glands, pleura, spleen, liver, mesenteric lymph-nodes, intestines, peritoneum, meninges and brain.

DIAGNOSTIC AIDS

Diagnostic Tuberculin Tests.—The specificity of the tuberculin reaction is today undoubted. A minute quantity of tuberculin produces a specific reaction in most infected individuals, while in the non-tuberculous relatively large quantities are inert. Tuberculin tests are, therefore, a reliable aid in the diagnosis of tuberculosis in infants and young children. Infection with the tubercle bacillus produces antibody-like substances called *ergins*. When these come in contact with the bacillus or with tuberculin, toxic substances, called *apotoxins*, are formed. This occurs at the site of the tuberculin application (local reaction), also at the foci where tubercle bacilli have become established (focal reaction). Tuberculin sensitivity of the body is spoken of as *allergy;* when this sensitivity is reduced or lost, the condition is known as *anergy*. In an anergistic period, as, for example, during a course of measles, a latent tuberculosis may become active. During anergy a tuberculous process may be active in spite of repeated negative tuberculin tests. One may expect negative tuberculin reactions in tuberculous individuals during the so-called incubation period of tuberculosis, in acute tuberculous infection with severe constitutional symptoms (in military tuberculosis, tuberculous meningitis, and advanced pulmonary tuberculosis); also during and for some time after certain infectious diseases, such as measles (in 100 per cent.), scarlet fever (in 84 per cent.), diphtheria (in 12 per cent.). The tuberculin test has also been found negative in tuberculous individuals with lobar pneumonia, influenza, erysipelas, typhoid fever, and during or following tuberculin therapy. In tuberculous infants the first cutaneous test is not infrequently negative. In a recent series of 25 tuberculous infants, Finkelstein[25] found the first reaction negative in 12 per cent. of the cases.

While the great majority of tuberculous indivduals react to both human and bovine tuberculin, a number of reports have appeared where only the bovine tuberculin gave positive reactions. Thus Finkelstein[25] cites cases of tuberculosis which unquestionably reacted only to bovine tuberculin. Kleinschmidt[40] shows conclusively that tuberculin tests made with each type of tuberculin cannot be relied upon to differentiate the bovine from the human type of infection. Klose,[42] a short time ago, introduced a mixed tuberculin for diagnostic purposes prepared from both types of bacilli. The relationship of the two types of bacilli is so close that most patients infected with the bovine type will also react to tuberculin of the human type.

The four diagnostic tuberculin tests which are at present most in use in determining tuberculous infection are:

1. The cutaneous test of v. Pirquet.
2. The intracutaneous test of Mantoux.
3. The subcutaneous (local) test of Hamburger.
4. The percutaneous test of Moro.

The second and third of these tests are by far the most sensitive and reliable, but more complex, cumbersome and painful. Their greater sensitiveness is probably due to the larger area in which the tuberculin and ergins of the skin react. Careful observation has shown that any of these tests, properly performed, will not reactivate old, quiescent lesions. Their application seldom produces a so-called general reaction—fever, malaise, etc.—the effect of an excess of produced apotoxin.

The severity of the reaction has little bearing on the activity of the process. It is quite certain, however, that the amount of ergins (antibodies) is most abundant during the first few years after an infection with the bacillus, during a recrudescence, and after a re-infection. For this reason, an intense reaction may signify that a focus has been active relatively recently. It need not imply that the disease is progressing, as it may be receding at the time the test is made. It is quite possible that the infection was limited to a few insignificant lymph glands. It must also be remembered that infected children with exudative diathesis usually give intense reactions. The history, age of the patient, clinical picture, etc., must be relied on in deciphering the significance of a positive reaction.

The Cutaneous Tuberculin Test (**v. Pirquet**).—The cutaneous tuberculin test, introduced by v. Pirquet[67] in 1907, is the test of choice for most practical purposes. A positive reaction signifies tuberculous infection, not necessarily tuberculous disease. Only in infants and very young children does it usually imply an active tuberculous process. Reuschel[73] and Hamburger[35] have conclusively shown that this test cannot be relied upon to determine the incidence of tuberculous infection. Results are dependent upon such circumstances as: The instrument used in making the scarification (Pirquet borer, scalpel, needle), the depth of the scarification, the skin turgor, the length of time the tuberculin is allowed to act, the activity of the tuberculin, the number of times the subject is tested, and the season of the year. Hamburger got 40 per cent. less positive reactions with the cutaneous than with his subcutaneous test. His cases gave a greater number of positive reactions in the spring than in the autumn. A negative cutaneous test, therefore, does not absolutely exclude tuberculosis. Von Pirquet,[68] Feer,[24] Finkelstein[25] and others recommend repeating the test on the same arm a week later if the first test is doubtful or negative. The regional reactivity of the body is thus often increased. Not infrequently the repeated test gives a positive reaction. Tuberculous areas of skin react much more violently to the cutaneous test than does normal skin. If the second cutaneous test is negative, the intracutaneous or subcutaneous test may be resorted to.

Bass,[5] repeating cutaneous and intracutaneous tests in over two hundred infants and young children of an orphanage, got the following interesting results:

TABLE 3

	Number of positive cases	Percentage positive
One cutaneous test	7	3.3
Two cutaneous tests	14	6.8
Three cutaneous tests	21	10.1
Three cutaneous and one intracutaneous tests (0.1 mg)	34	16.5
Three cutaneous and two intracutaneous tests (0.1 mg.; 1 mg.)	47	22.8
Three cutaneous and three intracutaneous tests (0.1 mg.; 1 mg.; 1 mg.)	52	25.2

This again shows most conclusively that one negative cutaneous tuberculin test need not exclude tuberculous infection. Whenever tuberculosis is suspected a negative cutaneous test should be repeated or one of the more accurate tuberculin tests should be employed.

E. Schloss[76] working with older children got similar results:

Fourty-five per cent. gave a positive cutaneous reaction the first time.

Twenty-five per cent. of those who failed to react gave a positive reaction on repetition.

Fifteen per cent. of those remaining negative, gave a positive reaction with the intracutaneous test.

Technique of the Cutaneous Test.—The skin of the flexor surface of the left forearm is wiped off with a pledget of sterile cotton saturated with ether. When dry, two drops of undiluted Old Tuberculin are placed on the skin 10 cm. apart. Using a twisting motion with a sterilized (flamed) Pirquet borer a control mark is made midway between the two drops. Bleeding must be avoided. The sacrification should be made as round as possible. A similar mark is made through each of the drops. After five minutes the excess of tuberculin is carefuly wiped off. If desired, a sterile gauze dressing may then be applied.

If possible, the test should be inspected at the end of 24 hours; otherwise, surely at the end of 48 hours. The specific reaction is usually a red, elevated, firm papule, more than 5 mm. in diameter.

The various types of positive reaction which have been observed are:

1. The *usual reaction*—appears within six hours and begins to fade on the second day Plate I.

2. The *premature reaction*—rapid evolution of slight intensity, reaches the maximum within 10 hours and disappears on the second day.

3. The *persistent reaction*—reaches its maximum slowly, not within 48 hours and does not fade within a week.

4. The *late reaction*—appears after 24 hours and develops and recedes slowly.

5. The *cachectic reaction*—infiltration but no redness.

6. The *scrofulous reaction*—small, elevated nodules, surrounded by a tender, hyperemic area.

The reaction rarely becomes vesicular and very rarely pustular or hemorrhagic. *Doubtful reactions* have a diameter of less than 5 mm. and *secondary reactions* are those that are positive when performed a second time. Doubtful and secondary reactions, as well as those which are elicited by intra- or subcutaneous tests after two negative cutaneous tests probably signify that the body has been infected but is not making many ergins at the time.

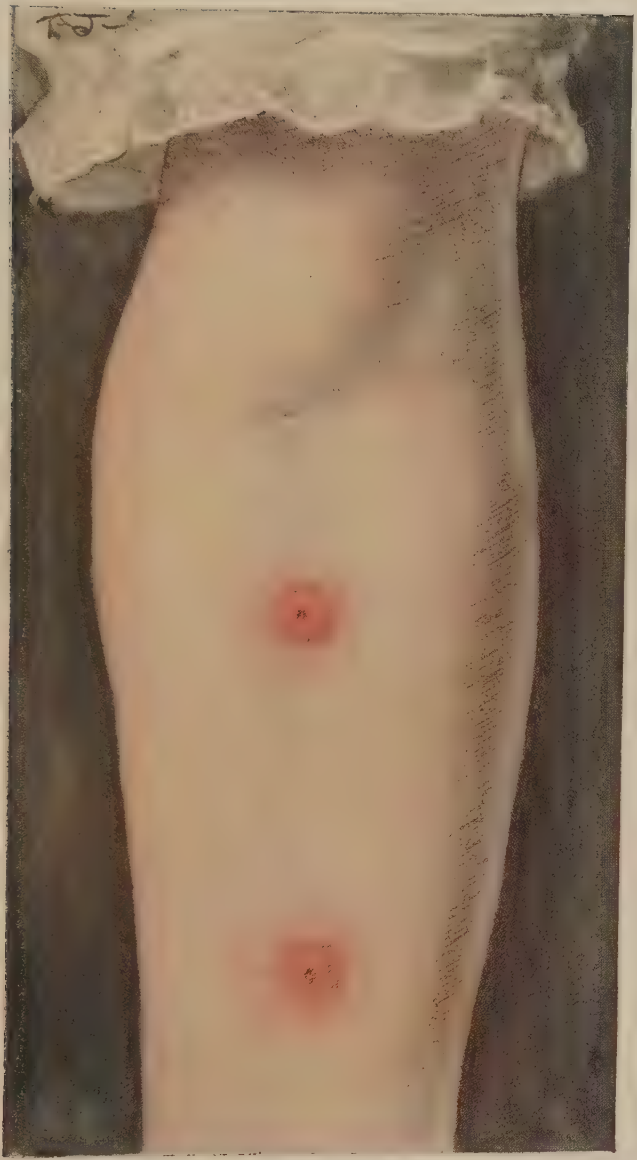
Moro[55] says a positive cutaneous test in a child under two years of age is absolute proof of the presence of active tuberculosis, because inactive tuberculosis practically never occurs in such young children. The younger the child the more significant a positive reaction, the older the child the more significant a negative one.

Feer,[24] Conquist,[17] and others maintain that a positive cutaneous reaction becomes negative when a tuberculous process has completely healed.

Intracutaneous Test (Mantoux).—This test[53] is more exact, reliable and sensitive than the cutaneous. Only by the exercise of extreme precautions can satisfactory results be obtained. A certain number of advanced as well as incipient cases will give a positive reaction with this test, whereas the cutaneous test will fail to detect the disease, and by its use the so-called incubation period is shortened by two or more weeks. Its main field of application is in hospital cases when the incidence of tuberculous infection is studied, or when several cutaneous tests are negative. Positive reactions are sharply defined.

Technique.—The freshly diluted Old Tuberculin is injected between the layers of the skin, producing a white wheal. A control injection of sterile normal salt solution should also be made. The concentrations now in vogue are: 0.01 mg. or 0.1 mg. Old Tuberculin in 0.1 c.c. of sterile, normal salt solution. Although stronger solutions are seldom necessary, advanced cases may require as much as 1.0 mg. A positive reaction consists of infiltration and hyperemia within 24 to 48 hours which remains more or less visible and palpable for nearly a week. A negative reaction as well as the control may show a traumatic hyperemia which disappears within 48 hours. The diameter of the infiltration in positive reactions should measure at least 5 mm. and

PLATE I



Positive cutaneous reactions. The control scarification is scarcely visible.

readings should be made at the end of 48 hours. A negative reaction in advanced clinical tuberculosis signifies that ergins are absent.

Subcutaneous (Local) Test (Hamburger Stichreaktion).—Due to the difficulties encountered in the performance of this valuable test,[34] its application is similar to that of the intracutaneous test. According to Finkelstein[25] atypical reactions are not infrequent, such as: Edema without redness, transient swelling of short duration, and diffuse swelling and redness extending beyond the area of injection. Mild constitutional symptoms may accompany a positive reaction.

Technique.—An injection of 0.01 mg. of freshly diluted Old Tuberculin is carefully introduced into the subcutaneous tissue immediately under the skin. If the reaction is positive, redness and infiltration begin about the site of injection within 8 to 12 hours. Those who do not react in 48 hours should receive 0.1 mg. in the same way. If this is negative in 48 hours, 1.0 mg. is given. If the site of injection does not show a positive reaction, the child may be regarded as free from tuberculosis with nearly absolute certainty. In doubtful cases 10.0 mg. may be injected.

Percutaneous Test (Moro).—The percutaneous test[55] is slightly less sensitive than the cutaneous, and may be resorted to in those cases where the parents resent the latter. It has been serviceable also in determining the result of treatment. The more marked the reaction, the better the prognosis. The absence of a reaction in active tuberculosis has been interpreted as unfavorable. In scrofulous children the reaction is sometimes very severe and extensive.

Technique.—Five cubic centimeters of Old Tuberculin and 5 gm. of anhydrous lanolin are well mixed and an amount the size of a small pea is rubbed for one minute into the skin of the upper abdomen or chest over an area 5 cm. in diameter. The usual positive reaction consists of small red papules on the anointed region within 12 to 48 hours. Late as well as disseminated reactions are occasionally encountered. Moro describes three degrees of reaction—mild, moderate and severe. Some clinicians (*e.g.*, Feer) have simplified the technique by merely dropping undiluted Old Tuberculin on an area of the skin and rubbing it in for a minute. Widowitz[85] produces a hyperemia of the sternum with a pledget of cotton containing ether and then a drop of tuberculin, which has been concentrated on the water-bath, is well rubbed in. The reactions are similar to those above described.

Complement-fixation Test.—The results of the complement fixation test in the diagnosis of tuberculosis are somewhat confusing. Punch[70] and others conclude that the reaction is specific for active or recently active tuberculosis. Von Wedel[82] and others found that many non-tuberculous cases gave a positive reaction, while in a certain proportion of tuberculous cases it was negative. Petroff[64] claims the test to be more specific for tuberculosis than the Wassermann test for syphilis. It should be considered only one of the many links in the tuberculosis diagnostic chain. The technique is not yet standardized and, in order to obtain reliable results, the test should be performed only by well trained workers.

Presence of the Bacillus.—Finding the bacillus in sputum, stomach contents, spinal fluid pellicle, exudate, sinus, gland or ear discharge, urine, stool, or skin lesion is absolute proof of tuberculous disease. As children seldom expectorate before the seventh year, the best way to get sputum is to aspirate the stomach after fasting or after a severe coughing spell, or to gag the child with a swab. It is advisable not to make a positive diagnosis unless a number of typical tubercle bacilli are demonstrated in a preparation. Organisms of similar morphology and acid-fastness (B. smegma and B. lepræ) must be excluded.

Guinea-pig inoculation is the most delicate test for demonstrating the presence of tubercle bacilli. Such inoculation of pleural exudate, urine, etc., is a valuable diagnostic acid, but the subsequent lesions must prove to be indisputably tuberculous.

X-ray.—Although the *x*-ray is a valuable aid in diagnosis, we have learned that it often fails where it is most needed—in the early diagnosis of chest conditions, especially of bronchial gland tuberculosis in infants and young children. An experienced and critical eye is necessary, and constant recourse to the clinical picture must be made; and even then, error is not uncommon. Only clear-cut, symmetrical pictures are of value and exposures must be very short. Stereoscopic pictures may show more definitely the topography and extent of the lesion.

A shadow corresponding to an enlarged bronchial gland must correspond topographically to that gland. The sources of error are numerous. Indefinite shadows leave much room for conjecture. The primary focus, a bean-sized, dense, sharply demarcated, isolated shadow, free in the lung area beyond the hilus, with an enlargement at the same time of the regional glands, may be detected in early cases with a positive tuberculin test (Plate I). A positive diagnosis of bronchial gland tuberculosis is difficult and can rarely be made by *x*-ray. Negative findings need not exclude enlargement of the bronchial glands, because the enlarged glands may be in the median shadow; nor do enlarged glands necessarily imply tuberculosis, as other conditions may cause such pathology.

Tuberculous infiltration of the lung may appear as dense or hazy shadows; cavities as more or less sharply circumscribed light areas; early miliary dissemination as fine "snow flakes" (Fig. 336); advanced miliary dissemination as coarse "snow flakes" (Fig. 337). Interlobar adhesions can be detected as longitudinal shadows, corresponding to the border of the lobes. Pictures repeated at intervals may aid in drawing conclusions. A hilus shadow will, in the course of months, become more dense, and narrow streaks will radiate into the lung tissue; or new, isolated shadows will appear; or the shadow appears smaller, the cardiac outline clearer, and the cardiodiaphragmatic angle will again be visible.

COURSE OF TUBERCULOSIS

Primary Lung Focus.—The evolution of the pathological evidence which led up to the acceptance of the primary lung focus is of interest. In 1876 Parrot[63] enunciated his law: "There is no affection of the lungs which is not also present in the adjoining lymphatic glands; and, *vice versa*, no changes occur in the tracheo-bronchial lymph glands without analogous ones in the lung." Cornet[16] showed that the regional lymph glands enlarge where the infection enters. He found the primary lung focus in 145 cases of tuberculosis. In 1898 Küss[49] said: "The inhalation of the bacillus explains this form of tuberculosis, characterized by a small tubercle, usually situated subpleurally. The lymphatic glands adjoining the lung focus nearly always become tuberculous—evidence of a connection between the seat of the primary lung focus and that of the altered lymphatic gland. The size of the primary lung focus varies from that of a pin's head to that of a hazelnut." E. Albrecht[62] pointed out in 1906 that one can usually discover a macroscopic focus in the adjacent lung or lobe of the lung exactly corresponding to the affected regional lymph glands. H. Albrecht[3] states that the primary lung infection is represented by an isolated focus often found caseous, frequently containing in its center a thin fissure, the remnant of a bronchiolus. There is usually only one, but there may be several foci in one or both lungs. He shows the true site of the primary infection to be in the smallest lymphatic glands of the finer bronchi, and concludes that the primary tuberculous infection is aerogenous. Naegeli[57] maintained that only a pulmonary origin could be assigned to a tuberculosis of the bronchial glands, with the primary focus in the lung. If the primary lung focus cannot be found, the germ passed through the lung without leaving a lesion.

Ghon's results,[29] published in 1912, confirm the evidence of Küss and H. Albrecht. He considers the aerogenous route the most frequent, resulting in a primary lesion in the lung, with secondary lymph gland involvement. As proof against the hematogenous route of infection in the origin of the primary lung focus, he points out that in 83.5 per cent. of his 184 cases, only one lesion was present, instead of the multiple lesions which one invariably finds in metastatic processes. In 38 of the 58 necropsies studied by H. Koch[43] there was a single primary lesion, in eight there were two primary lesions, in four he found three and in one child four lesions. In the remaining eight cases widespread tuberculosis had involved the entire lobe in which the lesion occurred. In only three of the 58 cases was there a sign of healing.

In 95 per cent. of the children who have a tuberculous lesion, the primary focus is in the lung and the secondary one in the bronchial lymph glands. In the other 5 per cent. the primary lesion is in the alimentary tract, on the skin or genitals.

The course of tuberculosis, according to Hamburger,[33] is similar to that of syphilis with regional adenopathy, immunity against re-infection, periods of health and latency interrupted or followed by periods of acute or subacute exacerbations. The primary lesion usually occurs before the tenth year of life. If massive infection takes place or if the resistance of the host is low, the process may go on to miliary tuberculosis or hematogenous metastases. In the vast majority of children after infancy it heals or remains dormant.

By **incubation period** of tuberculosis is understood the time elapsing between infection and the first positive tuberculin test, or until the first clinical symptoms are observed. H. Koch[43] used the cutaneous test and found it to be seven to eight weeks. Hamburger[33] using his subcutaneous test, found it to be about 30 days. The period is found shortest in the newborn, when the infection is massive and when the subcutaneous or intradermal test is used.

The **primary stage,** according to Hamburger,[33] Petruschky,[65] and v. Pirquet,[68] is reckoned from the first occurrence of a positive tuberculin test until the process has spread to the regional lymph glands. The course of the disease will end here if the developed immunity suffices to protect the individual.

The **secondary stage** is the period of hematogenous or lymphogenous distribution into the various organs beyond or outside of the regional lymph glands, producing tuberculosis of the lungs, glands, bones, joints, skin or meninges. Most of the various types of clinical tuberculosis of childhood belong here.

The **tertiary stage** usually occurs after the tenth year (during puberty or later) in the various forms of chronic phthisis, tuberculosis of larynx, kidney, adhesive pleurisy and lupus. It is the result of a complete collapse of the protective mechanism of the body.

GENERALIZED TUBERCULOSIS

Generalized or general tuberculosis includes infantile tuberculosis (the pre-natal and early post-natal types) and miliary tuberculosis.

Etiology.—The younger the child the less likely will the body produce sufficient immunity to inhibit the progress of the infection. The age of the host, and the size and virulence of the primary infection are factors which determine whether the disease will remain limited to the regional lymph glands or whether extensive destruction of contiguous parts with subsequent dissemination by lymph and blood streams will occur. As the primary lesion in the lung and the infection in the regional lymph glands grow, the greater part of a lobe may be destroyed by caseation, softening and central cavity formation; a caseous gland may break into a vein, whereby many bacilli are carried to other organs and metastatic foci develop. If the necrotic area in the lung communicates with a bronchus, a tuberculous aspiration pneumonia may result. Swallowed bacillary sputum may produce ulcers in the alimentary canal. Buhl in 1856 said that miliary tuberculosis depends on the presence of a tubercle in the body or a tuberculous cavity in the lung and likened miliary tuberculous dissemination to the spread of pyemia from a primary focus of suppuration. The disseminated bacilli do not multiply in the blood, but lodge in the various organs, and generalized tuberculosis results.

Pathology.—Regardless of the portal of entry and whether the infection occurs shortly before or soon after birth, the disease develops rapidly in the various parts of the body. At necropsy miliary, submiliary, or nodular lesions are found in various organs (lungs, liver, spleen). Signs of healing are nearly always absent. In the prenatal type the primary focus is absent, but lymph glands in various parts of the body are frequently enlarged. The spleen and liver are always involved and contain small and large tubercles. If the disease was contracted after birth the lungs and bronchial glands usually show the most extensive involvement. A lobe or an entire lung (Fig. 339) may be caseous. A cavity may be present in the center of the caseous area. Fibrous pleurisy is usually extensive.

PRE-NATAL (CONGENITAL) TUBERCULOSIS

Symptoms and Physical Signs.—The child is usually underweight and soon appears atrophic. Cough may be present, but is seldom conspicuous. The liver and spleen are much enlarged and the abdomen is more or less prominent. Tuberculides may be present. Examination of the lungs may reveal small areas of dulness and bronchial breathing. At times fluid may be detected in the chest.

Diagnosis.—A history of poor health on the part of the mother is very important. The child fails to thrive. Tuberculides may be present. The diagnostic tuberculin test is positive before the child is a month old. An *x*-ray examination may show areas of consolidation in one or both lungs. Chronic bronchitis, pneumonia, sepsis and emphysema must be excluded.

Prognosis.—The disease is usually fatal within a few weeks or months after birth.

Treatment.—Isolation to avoid super-infection, breast milk from a non-tuberculous source, fresh air and symptomatic treatment should be faithfully persisted in.

EARLY POST-NATAL TUBERCULOSIS

Symptoms.—There is usually nothing characteristic of the disease. The general appearance of the infant is frequently that of a mild atrophy. Fever, if present, is often intermittent; when persistent, the weight loss is usually rapid. The appetite is invariably poor. There may be a dry, hacking cough often simulating that of pertussis. Respirations are usually somewhat accelerated and a slight degree of cyanosis may be present.

Physical Signs.—Percussion of the chest may reveal nothing or there may be dulness over an area of the lung. Auscultation may, at times, detect areas of diminished or bronchial breathing. Cavities may be detected shortly before the end or necropsy may fail to reveal those surmised during life. The spleen and liver are distinctly enlarged. There may be a chronic otitis media. The urine or aspirated sputum may contain tubercle bacilli. The tuberculin test is usually positive.

Diagnosis.—A suspicious family history or other history of exposure is very important. If repeated *x*-ray pictures show a cavity in the lung of an infant with a positive tuberculin test the diagnosis of general tuberculosis is very probable Finding the bacilli makes the diagnosis certain. Typhoid fever, chronic bronchitis, chronic pneumonia and empyema must be ruled out. Not infrequently the diagnosis is first made at necropsy.

Prognosis.—The prognosis is bad but the advent of the diagnostic tuberculin test leaves no doubt that some cases recover. The course is variable. Recovery may require many months.

Treatment.—Isolation to prevent super-infection, fresh air and proper food are the chief therapeutic measures at our disposal. Gavage feeding may be necessary.

Acute Miliary Tuberculosis

The onset of miliary tuberculosis is only occasionally recognized, as the early symptoms are rather vague: Anorexia, irregular fever, and progressive loss of weight without apparent cause. A history of exposure and a positive tuberculin reaction are valuable aids in diagnosis. The symptoms may, for a time, simulate more or less those of typhoid fever. The fever may increase, but remains irregular with the highest peak in the evening. Some cases may run an afebrile course for weeks. After some time the respiratory rate usually increases and signs of a localized or generalized bronchitis become manifest. Cerebral symptoms such as headache, vomiting, irregular pulse, irregular respiration of the Cheyne-Stokes type, convulsions and coma frequently develop toward the end, and imply meningeal or cerebral involvement. At times the pulmonary condition spreads rapidly and dulness and tubular breathing become definite and may involve an entire lobe or lung. In older children the course may be more protracted, and after some weeks a tuberculous bronchopneumonia may hasten the end. If meningeal symptoms predominate the course will be that of a tuberculous meningitis.

Diagnosis.—Fever, when present, is usually much more irregular than in typhoid fever; diarrhea is less likely and rose spots are absent. The presence of tuberculides is of great diagnostic value. Leukopenia is absent in tuberculosis. Choroidal tubercles, tubercle bacilli in sputum, urine, spinal fluid or blood culture, a positive tuberculin reaction and an *x*-ray picture showing the characteristic stippling of the lungs make the diagnosis certain (Fig. 336).

Prognosis.—The course is usually steadily downward, as miliary tuberculosis in infants and young children usually ends fatally within six weeks. The prognosis is not as hopeless as was once thought.

If only a scant dissemination has occurred in a child past infancy, recovery is not impossible.

Treatment.—The treatment is symptomatic and constitutional. Tuberculin therapy is contra-indicated. Infants should receive breast milk from a non-tuberculous source. Signs of intracranial pressure must be relieved. Sedatives, cathartics, cardiac and respiratory stimulants should be resorted to when indicated.

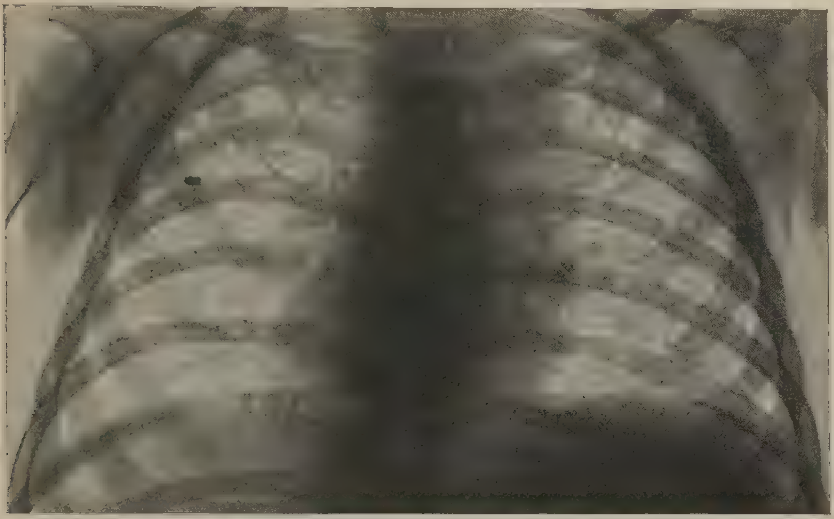


FIG. 336.—Acute miliary tuberculosis of the lungs. W. D., two years old. Symptoms typical, but few physical signs. Temperature varied between 100 and 102°F. Cutaneous tuberculin test positive; *x*-ray of chest shows fine "snowflakes"—shortly after dissemination; thymus rather large. Died of tuberculous meningitis 12 days later.

PULMONARY TUBERCULOSIS

Pulmonary tuberculosis usually results from the growth of the primary focus in the lung, the aspiration of caseous material from a softened regional lymph gland which has ruptured into a bronchus, or the spread of a tuberculous lymph gland to neighboring parts of the lung.

A diagnosis of active tuberculosis in infants and children is usually made after the protective mechanism of the body has been destroyed, when marked destruction of vital organs has already occurred. While phthisis, as seen in adults, may occur at any age, it is relatively rare before puberty.

The following clinical forms of pulmonary tuberculosis vary sufficiently to deserve separate descriptions.

Miliary Tuberculosis of the Lungs.—The general and pulmonary signs and symptoms may be obscure. The weight loss may be gradual until shortly before death, or, as is more frequently the case, the child may appear severely ill with marked dyspnea and cyanosis as in a capillary bronchitis or acute bronchopneumonia. There may be a cough and râles, but the auscultatory and percussion findings are insufficient to explain the dyspnea and cyanosis. The fever is persistent, irregular, and seldom very high until shortly before the end. As time goes on, the respiratory rate increases, liver and spleen become palpable. Symptoms of meningitis may occur. The x-ray usually reveals a multitude of miliary tubercles scattered throughout both lungs (Fig. 337). The disease usually ends fatally in two to six weeks. The cases of recovery reported in the literature were probably those where small miliary showers occurred in the course of a recent tuberculosis. Therapy is chiefly symptomatic. Oxygen may be of some value in the relief of cyanosis. The treatment outlined under "Miliary Tuberculosis" should be followed.

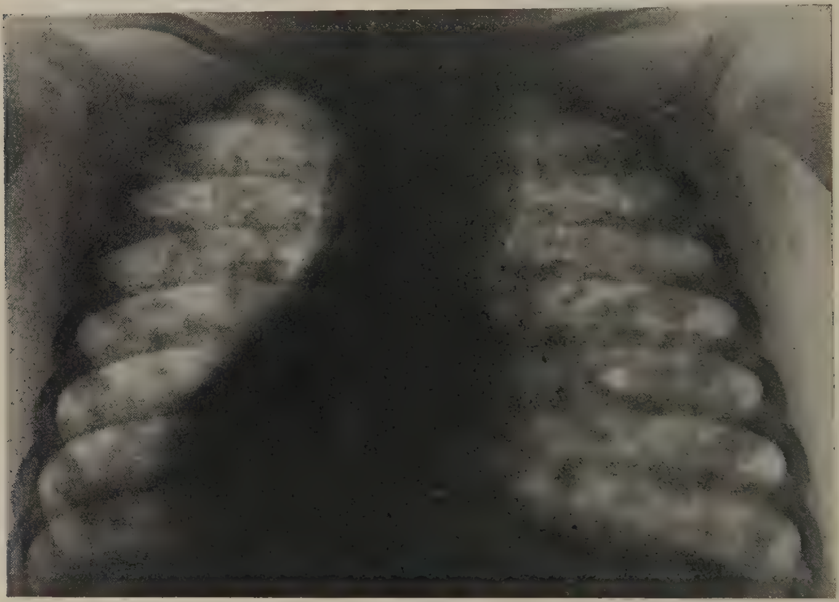


Fig. 337.—Lung in miliary tuberculosis. Francis G., five and one-half months old. Irregular fever for several weeks, labored respiration for several days preceding admission. Cutaneous tuberculin test positive. X-ray shows coarse "snow-flakes"—growth and coalescence of milary tubercles. Death a fortnight later with symptoms of tuberculous meningitis. Diagnosis substantiated by necropsy.

Tuberculous Bronchitis.—Tuberculous bronchitis is usually a transient condition of the lungs, occurring most frequently in children under five years of age. The lungs become involved by contiguity from tuberculous bronchial glands, a primary focus may spread or a caseous gland may rupture into a bronchus.

Pathology.—The pathological findings are those of a more or less diffuse bronchitis with tubercles scattered through the lung tissue and bronchi. The bronchial glands are always involved.

Symptoms.—The symptoms may be few and differ in no respect from those of an ordinary bronchitis. The general condition of the patient is not infrequently poor. There is more or less wheezing in the chest, the cough is persistent, dry and non-productive. An irregular fever soon develops, and persists for weeks or months.

Physical Signs.—The physical signs are also more or less inconclusive. Coarse and fine râles are more or less constantly present, and are usually localized in the third and fourth intercostal spaces to either side of the sternum.

Diagnosis.—A positive tuberculin reaction in a young child with the above findings should arouse suspicion. An *x*-ray may show enlarged bronchial glands or hilus infiltration. Finding tubercle bacilli in the sputum makes the diagnosis certain, although it may signify that the condition has already advanced to a tuberculous bronchopneumonia.

Tuberculous Bronchopneumonia.—Tuberculous bronchopneumonia (caseous pneumonia) is the most common form of pulmonary tuberculosis occurring in infancy and childhood.

Etiology.—The condition may be apparently primary or it may occur in the course of a chronic tuberculosis elsewhere, especially in the bronchial glands. It is not infrequently a sequel to one of the acute infectious diseases, such as measles, pertussis and influenza, or may apparently follow a chronic bronchitis or a catarrhal bronchopneumonia.

Pathology.—The caseous areas found at necropsy vary greatly in size and number. They are either limited to one lobe or scattered throughout one or both lungs. In the former case, an upper lobe is not infrequently involved. Caseation may involve an entire lobe or lung (Fig. 338). The visceral pleura is usually thickened and the lung is frequently adherent to the parietal pleura and chest wall. Cavities are usually small, but sometimes multiple. The bronchial glands are usually more or less caseous. Serous or purulent exudate and tuberculous lesions in other organs are not infrequent.

Symptoms.—When the onset is sudden, as in the acute form, the symptoms may be those of an ordinary pneumonia. Cough, rapid respiration and asthenia are then very pronounced. When the onset is more gradual the cough is deep, dry, and more or less suppressed and non-productive. The irregular fever is not high but constant. Lack of appetite, anemia, apathy and loss of weight increase. A week or more before the end the dyspnea, cyanosis, feeble pulse and prostration become pronounced. The cough may be paroxysmal, somewhat resembling pertussis. Cerebral symptoms such as convulsions, irregular respirations, rigidity and strabismus denote involvement of the central nervous system or meninges.

Physical Signs.—There may be almost no signs in the lungs or the findings may be insufficient to explain the marked asthenia of the patient. Consolidation and bronchial breathing may be absent for weeks, or the patient may die before signs of consolidation can be elicited. The right upper lobe is the site of predilection in infants and young children. The spleen is usually palpable. Fine or coarse râles may be scattered throughout both lungs. Cavities are seldom detected during life without the *x*-ray (Fig. 339).

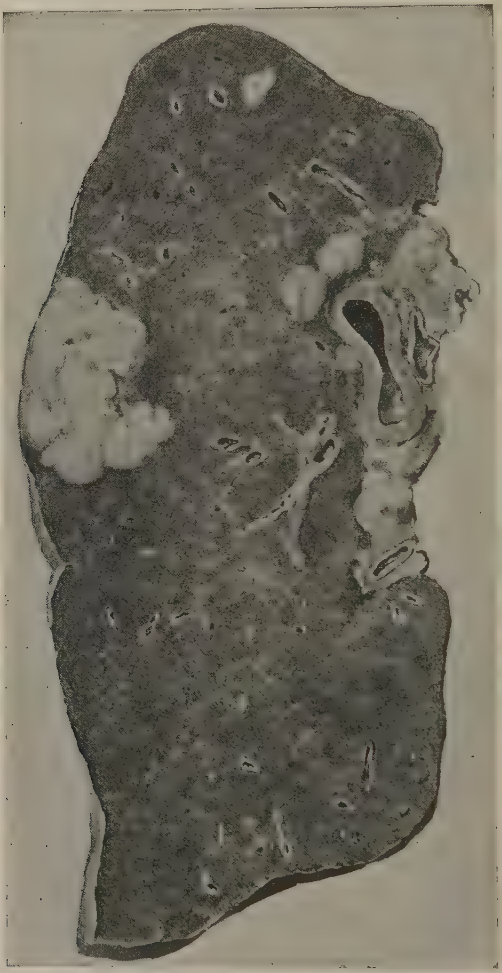


Fig. 338.—Tuberculous bronchopneumonia, caseous primary focus and bronchial glands (longitudinal section).

Diagnosis.—The presence of an open case of tuberculosis in the household or slow recovery from one of the acute infectious diseases is suggestive. Accelerated respirations, out of proportion to the fever and physical findings, in an infant or young child with a positive tuberculin test should make one suspect tuberculous bronchopneumonia, even when the chest findings are indefinite or absent. The *x*-ray may reveal areas of consolidation or enlarged bronchial glands which may be considered further evidence. Finding the bacilli in the sputum or fasting stomach contents makes the diagnosis certain.

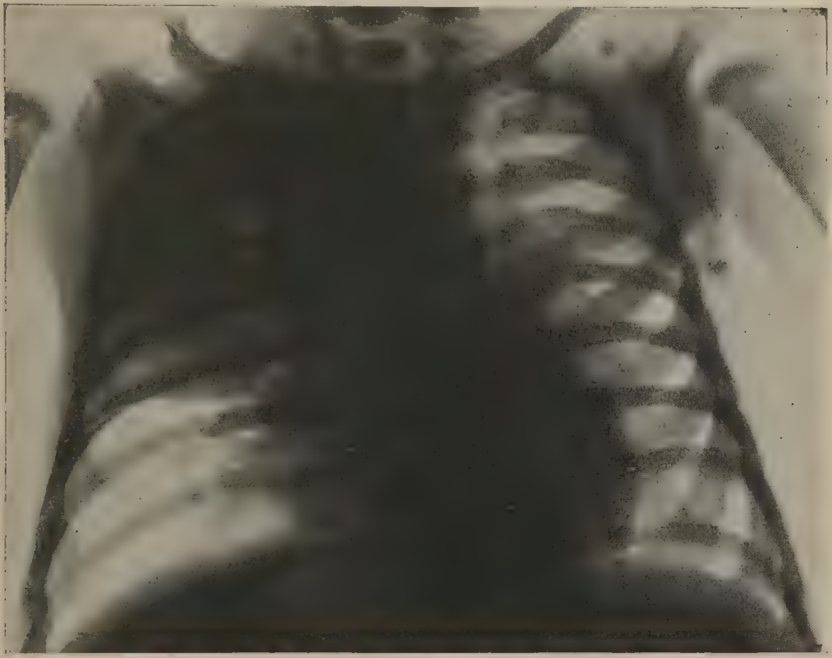


FIG. 339.—Tuberculous bronchopneumonia (shows cavity and pneumothorax). Casimer O., four and one-half months old when admitted. Cough for three months with rapid loss of weight; physical signs those of a bronchopneumonia involving the upper and middle lobes of the right lung. Sputum repeatedly negative; two cutaneous tuberculin tests negative. Diagnosis: tuberculous bronchopneumonia with cavity in center of consolidation and pneumothorax (substantiated by necropsy). Death three weeks after admission.

Prognosis.—The course is usually steadily downward, and in children under three years ends fatally in 3 to 12 weeks. In exceptional cases partial resolution of the pneumonic process occurs and a chronic fibroid pneumonia results. In the course of time there may be a fatal exacerbation of the pulmonary condition, a miliary tuberculosis of the lungs, tuberculous meningitis or a terminal pneumococcic bronchopneumonia. The prognosis is always grave, but the older the child the longer the course and better the outlook.

Treatment.—Fresh, moving air, ample food and symptomatic therapy are the only therapeutic measures at our disposal in the treatment of this rather frequent and usually fatal form of tuberculosis.

Hilus Tuberculosis.—Hilus tuberculosis of the lung usually has its origin in the transgression of a hilus gland tuberculosis to the contiguous lung tissue.

Symptoms are vague. The onset is more or less stealthy with cough, anorexia, fatigue, slow loss in weight and an evening rise of temperature. The tuberculin reaction is positive, but physical examination may reveal very little or nothing. D'Espine's[20, 21] sign is usually present. Dulness to either side of the vertebræ is suggestive.

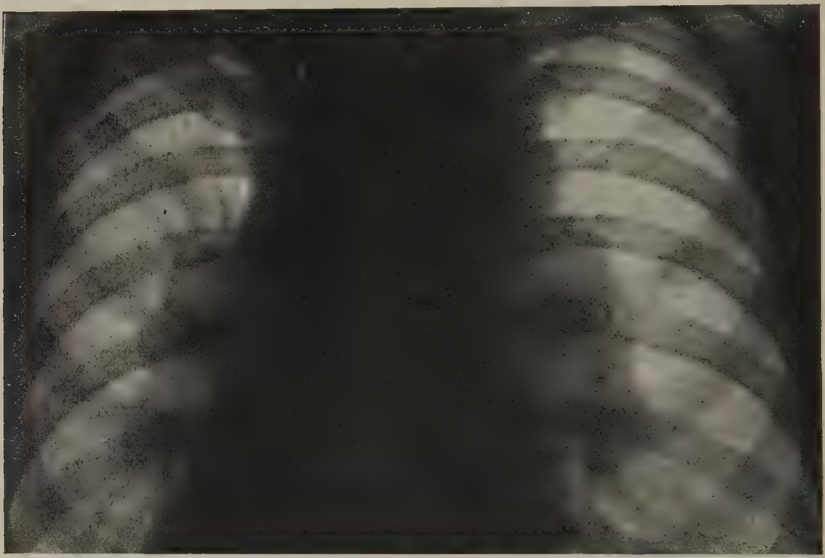


Fig. 340.—Hilus tuberculosis. Frank P., 16 years old. About a year before admission, the cervical glands slowly enlarged and the cutaneous tuberculin test was positive. Respirations are somewhat labored as in bronchial gland tuberculosis. D'Espine's sign is positive. The *x*-ray shows hilus involvement.

The *x*-ray usually shows a fan-like shadow radiating from the hilus (Fig. 340). A definite hilus shadow may be the source of confusion. A dense triangular shadow reaching into the lower lobe frequently seen on the right side need not imply hilus tuberculosis. Conclusive evidence must come from the general condition and presence of fever. After a number of months bacilli may be discovered in the sputum. This usually signifies advance of the process to other parts of the lung.

The course is that of a severe bronchial gland tuberculosis or the condition advances to further lung involvement.

Chronic Ulcerative Tuberculosis of the Lungs (Phthisis).—This form of pulmonary tuberculosis, so prevalent in adult life, occurs much more frequently at puberty than at any other period of childhood.

Etiology.—Von Behring[8, 9, 10] held that phthisis resulted from a re-infection in a person infected with the bacillus during early childhood. "Phthisis," he said "is but the last verse of a song, the first verse of which was sung at the cradle." Röemer[74] held that childhood infection with the bacillus, when not fatal, leads to an increased resistance against further development of the disease. Such an acquired immunity usually suffices to protect against exogenous re-infection for the rest of the individual's life. Should the resistance be lowered by special circumstances such as puberty or intercurrent disease, the bacilli in the body may multiply so rapidly that the existing immunity is insufficient, and new foci develop with clinical manifestations, often of a phthisical nature. Orth[59] has furnished pathological proof that old lesions need not show recent activity and that an acute tuberculous re-infection may supplant a quiescent one and cause death. He maintains that a massive exogenous or small endogenous re-infection may break down an existing immunity sufficiently to cause progress of a tuberculous process. R. Koch[47] observed that exogenous re-infection has a tendency to heal and does not involve regional lymph-nodes as does a primary infection. Apical tuberculosis, therefore, does not exhibit this characteristic of a primary infection. Bacmeister[4] could produce typical, apical phthisis in rabbits only when a previous tuberculous infection has occurred somewhere in the animal. He found that primary lung foci, produced by intravenous injection, would enlarge and caseate as in infants and non-immune races, and concludes: "For the production of typical phthisis, a relative immunity of the body is necessary, and this is produced by an early primary infection."

By superinfection is usually meant a second infection from without at a time when the lesions produced by the first infection are still active. Re-infection implies a new infection from within (endogenous) or without (exogenous) when the lesions produced by the first are not active.

Pathology.—Although it is rarely possible to detect the relation of the tertiary to the secondary or primary stage of the disease, pathology and animal experiments lead us to conclude that pulmonary tuberculosis is usually the third and final stage of a process which had its origin earlier in life. The lesions show a wide variation, consisting mainly of nodular tubercles, diffuse infiltration, caseous masses, areas of bronchopneumonia, pleuritic, bronchial and bronchial gland involvement. Four types of ulcerative lesions exist:

1. A single tuberculous cavity, possibly the primary focus; this is most frequent in infants.
2. A large, multilocular cavity in an area of tuberculous bronchopneumonia—the most frequent type.
3. Small, multiple cavities scattered throughout a lobe or an entire lung; each surrounds a bronchus—the result of dissemination of material from a tuberculous bronchopneumonia.

4. Cavities in dense fibrous tissue containing dilated bronchi—most frequent in older children.

Symptoms.—The so-called pretuberculous symptoms are: Stationary or progressive loss of weight, lassitude, pallor, anorexia, vasomotor disturbances (cold hands and feet), a dry cough on deep inspiration and, most important of all, an afternoon or evening rise in temperature without apparent cause.

After months or one or more winters, the cough becomes more severe, at times paroxysmal, especially in the early morning; expectoration becomes more profuse, loss of weight and anemia more pronounced. Fishberg[26] considers excessive perspiration in the early morning, pallor in the morning and bright red spots on the cheeks, glistening eyes and a slight fever in the late afternoon suggestive symptoms. Early hemoptysis, an important diagnostic sign in the adult, is not common in children, even at puberty.

Physical Signs.—Light percussion may reveal apical infiltration—the first valuable sign. Increased resistance, prolonged, blowing expiration, and exaggerated voice sounds are also very significant. Comparative auscultation of the apices may reveal a shortened, feeble breath sound with bronchial breathing accompanied by râles. Tachycardia is characteristic of active tuberculosis of the lung. When present, a cavity in the lower lobe can be detected rather easily. Expectorated material, if from the lung, often contains bacilli. Swallowed sputum may lead to intestinal ulcers which cause obstinate diarrhea or to laryngeal tuberculosis accompanied by chronic hoarseness.

Diagnosis.—A fluoroscopic examination of the chest will often reveal a diminished excursion of the diaphragm on the affected side, even before clouding of any portion of the lung occurs. Somewhat later the *x*-ray may show bronchial gland enlargement and definite areas of consolidation. The presence of tuberculides, chronic exudative peritonitis, Pott's disease, spina ventosa or other tuberculous conditions may be an aid in diagnosis. A positive tuberculin reaction in a child of more than five years is seldom of much diagnostic value. The sputum must be examined repeatedly for bacilli. The continued presence of large numbers of bacilli for weeks or months may signify that a cavity is either forming or is already present.

Prognosis.—The intrinsic tendency to heal is more pronounced in the adolescent than in the adult. The course is at least two years. In fatal cases death is due to exhaustion, or complications.

Treatment.—If the child is underweight, an increased diet and absolute rest should be instituted. If improvement sets in after a few weeks or months the child may be up for several hours of the day, even if there is a slight evening rise in temperature. The child should live out of doors whenever the weather permits, being dressed according to the season of the year. The extremities must be kept warm. Fresh air helps the appetite, and assimilation of food plays an important rôle in the cure of phthisis. For anorexia one may try five drops of creosote carbonate in sweetened milk twice a day. Cod-liver oil is of great value and, if tolerated, should be given routinely. If only one lung is involved, artificial pneumothorax may prove to be the best means of immobilizing the affected lung. The indications for this procedure are, however, more limited than in adults. If the pneumothorax is maintained (*x*-rays at weekly intervals), healing is greatly hastened. Eliasberg[23] gets the best results by the injection of filtered atmospheric air. It is not unlikely that harm may result from artificial pneumothorax in cases with bilateral lesions as the subsequent added activity of the untreated lung increases its blood supply and multiplies the chances of hematogenous dissemination of the bacilli. Atrificial pneumothorax should always be preceded by a thorough *x*-ray examination.

TUBERCULOSIS OF THE LYMPHATIC SYSTEM (SCROFULA)

Tuberculosis of the Lymph Glands

Tuberculosis of the regional lymph glands follows an infection somewhere within the domain of the diseased glands. The bronchial, cervical, and mesenteric glands are most frequently involved. Tuberculous lymph-adenitis tends to slow, spontaneous healing, except in early infancy. If suppuration occurs, the pus is usually sterile. Although tuberculous lymph-adenitis is a constant and serious menace (the potential source of active pulmonary, abdominal, meningeal, miliary, and bone and joint tuberculosis), the healed tuberculous process in childhood may confer subsequent immunity.

Generalized Tuberculosis of the Lymph Glands.—This rare form of tuberculous lymph-adenitis has been carefully studied by Leasage and Pascal.[51] It usually involves the various groups of glands successively, or, in exceptional instances, simultaneously. It is, at times, encountered in negro children or in those of other races which have but recently acquired the disease. Hodgkin's disease must be excluded. Death usually results from emaciation, meningitis, or an intercurrent disease. At necropsy the bronchial, mesenteric, and retroperitoneal lymph glands usually show marked enlargement with caseous degeneration.

Localized Tuberculosis of the Lymph Glands.—Localized lymph gland tuberculosis is one of the most frequent forms of tuberculosis in childhood.

Tuberculosis of the Bronchial Glands.—The lungs and trachea of infants and young children are extremely vulnerable to the tubercle bacillus, and the regional lymph glands become involved shortly after infection occurs. Although bronchial gland tuberculosis is one of the most common forms of the disease found at necropsy, its diagnosis during life is most difficult and uncertain. Its early recognition is of vast importance in the battle against tuberculosis in childhood.

Under *bronchial glands* are included all the lymph glands which accompany the air passages to the divisions of the large bronchi and those situated within the lungs. The glands which accompany the trachea and bronchi are divided into:

1. Those of the right and left tracheo-bronchial angle (*tracheo-bronchial glands*).

2. Those in the angle of divison into the two bronchi (*bifurcation glands*).

There are two groups of pulmonary glands:

1. Those which lie outside the lung in the angle of division of the first collateral branch into the lateral branch (*bronchopulmonary glands*).

2. Those within the lungs in the angle of division of the small bronchi (*pulmonary glands*). The latter two groups make up the group known as the *hilus glands*.

Topography.—Engel[22] points out that when interpreting an *x*-ray picture of the chest, the topography of the bronchial glands must always be borne in mind. The younger the child the more completely does the median shadow (vertebræ, heart and great vessels) cover the shadow of the bronchial glands. The *bifurcation glands*, the glands most frequently involved, always lie within the median shadow. The *right tracheo-bronchial glands* lie within the median shadow, very near the border or just outside the border. The *left tracheo-bronchial glands* lie within the shadow of the spinal column. The *right bronchopulmonary glands* in young children lie at the edge of the median shadow in the pulmonary field at a level with the junction of the upper and lower cardiac arches. The *left bronchopulmonary glands* lie within the median shadow at the upper end of the ventricular arch.

Pathology.—The disease is rarely limited to one group of glands or to one side, but the right glands are more extensively involved than the left (Fig. 341). They enlarge, caseate, and fuse into an irregular tumor which may occupy a large part of the mediastinun, pressing upon adjacent structures (nerves, vessels, trachea and bronchi). The right main bronchus may be compressed at the point between the bifurcation and the branching of the bronchus for the upper lobe. These glands are less likely to suppurate than are the cervical. In infants, calcification rarely occurs, and there is a tendency to the spread of the disease by ulceration and rupture into the air passages or blood-vessels.

Symptoms.—Extensive changes may be present in the glands and lungs without influencing the general condition, temperature and blood findings. There may be pallor, loss of weight, poor appetite, tendency to perspire and irregular fever. The most usual and constant symptoms are an obstinate, metallic bark, at times simulating pertussis, and a persistent hoarseness. An audible, expiratory dyspnea with prolonged expiration is also significant. The respiratory rate is seldom increased. Older children sometimes complain of pain between the scapulæ.

Physical Signs.—The thorax is often of the slender type. Pressure on the veins may cause those of the upper thorax and neck to be more or less prominent, the face may appear edematous, and cyanosis of the head and finger tips may be present. *Auscultation* over the dorsal spines may detect the presence of d'Espine's[20, 21] sign.

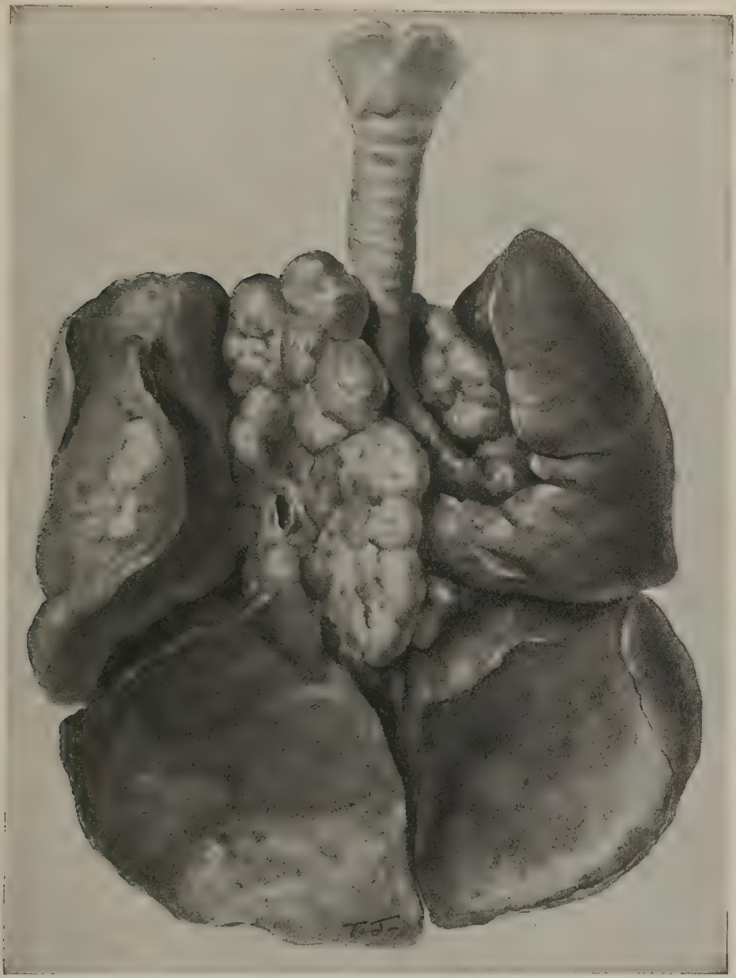


FIG. 341.—Tuberculosis of the bronchial glands. Barbara G. was admitted at the age of 14 months with a history of chronic severe, dry cough and fever. Cutaneous tuberculin test was positive and x-ray showed bronchial gland enlargement. The child died of a general tuberculosis a few weeks later. There were found: Caseous bronchial glands, bronchopneumonia of the middle lobe of the right lung, tuberculous caries of a dorsal vertebra and scattered tubercles in other organs, especially on meninges and brain. (Courtesy of Dr. H. Gideon Wells.)

The tracheal voice-sound should normally change into that of the lung in children under 10 years between the last cervical and first dorsal spines; in children from 10 to 12 years over the second, and in older children over the third dorsal spine. The presence of a spoken or whispered voice-sound extending lower than that found normal for a given age is known as a positive sign.

d'Espine's Sign.—It does not prove the tuberculous nature of the enlarged gland, but may serve as confirmatory evidence. A whispering sound heard after the spoken voice may also be present and is significant. *Percussion* is usually of little value. Dulness over the upper part of the sternum may be caused by the thymus; to the right of the sternum, to a tuberculous peribronchitis; between the scapulæ, to hilus tuberculosis. Dulness over the upper dorsal spines may be present, but is very difficult to detect.

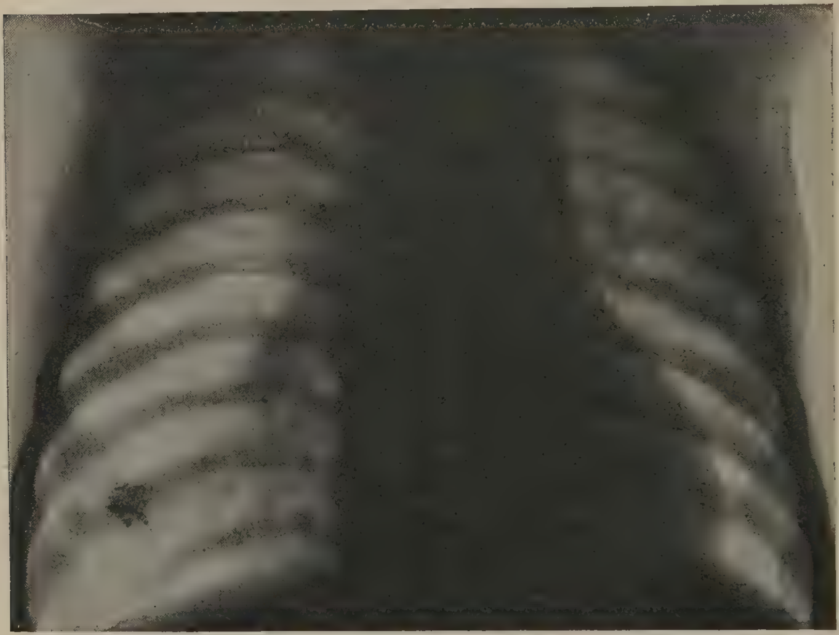


Fig. 342.—Primary lung focus and tuberculosis of bronchial glands. June P., eight months old. Father has a chronic cough. Child has been having expiratory dyspnea for two months. Cutaneous tuberculin reaction positive; breath sounds diminished over entire right side; *x*-ray shows only a little prominence at the right hilus. Primary lung focus is seen in the right lower lobe.

Diagnosis.—The *x*-ray may be a valuable but not conclusive aid in the diagnosis of bronchial gland tuberculosis (Fig. 342). Tuberculous bronchopulmonary glands of the right side which lie in the right pulmonary field may be typical. If the bronchial tree is not visible a bright line is present between the shadow of the glands and the cardiac shadow. Rach[71] says that in infants, the presence of enlarged tracheobronchial glands is the only pathological gland condition revealed by the *x*-ray. They produce a sharply demarcated convexity radiating downward from the mid-shadow parallel with the trachea. Diseased right hilus glands may be suspected when a triangular shadow is present, with its base at the margin of the heart and apex toward the periphery, but the clinical picture must decide whether or not a florid hilus phthisis does not already exist.

In older children a positive tuberculin test, suspicious *x*-ray findings and a positive d'Espine's sign may all be present in a receding or quiescent bronchial gland tuberculosis. In infants and young children such findings, especially when accompanied by expiratory dyspnea, make a diagnosis of active tuberculous bronchial glands almost certain. Due to difficulty in diagnosis, it is advisable to see the patient repeatedly before making a final diagnosis.

Bronchitis, bronchopneumonia, pertussis, and frequently syphilis, adenoid and tonsil hypertrophy, leukemia, and Hodgkin's disease cause enlargement of the bronchial glands. In infants a large thymus, and in older children a foreign body in a bronchus or lung may also produce symptoms and signs which may simulate tuberculous bronchial gland disease.

Prognosis.—The fate of the bacilli in these glands—their destruction or propagation—determines the future course of the disease. The younger the child the more serious the outlook. The course is always uncertain because of the numerous possibilities, among which must be mentioned ulceration of a pulmonary vessel with fatal hemorrhage or rupture of a caseous gland into a bronchus, causing asphyxia or disseminated tuberculosis. The course is usually benign in children over five years. Healing, if it occurs, is complete in 3 to 18 months.

Treatment. (See page 603 for a detailed discussion of treatment.)—Therapy is mainly along general lines—the child should receive sufficient rest and moderate exercise; sufficient and proper food, avoiding an excess of carbohydrates; fresh air and sunshine. A summer in the mountains or at the seashore or a winter in a dry subtropical climate is usually very beneficial. Before tuberculin therapy is resorted to pulmonary involvement must be excluded.

Tuberculosis of the Cervical Lymph Glands.—This is the most frequently diagnosed form of localized tuberculosis, probably because it is the most readily recognized. The primary seat of the infection is not infrequently the tonsil or pharynx. Mitchell[54] found the removed tonsils tuberculous in 41 out of 106 cases of cervical gland tuberculosis. Frazer[28] insists on the prevalence of the bovine type of bacillus in this lesion. Park and Krumwiede's[61] early work on the incidence of the human and bovine types of infection showed the former present in five, while the bovine type of bacillus was present in 46 cases. A similar investigation by the latter since the compulsory pasteurization of milk shows a much lower incidence of infection with the bovine type.[62]

The submaxillary glands of the affected side usually enlarge slowly. As they increase in size, the individual glands can usually be palpated for a time. The skin remains freely movable until pronounced inflammation and softening occur, when spontaneous rupture may follow.

After the pus has discharged the sinus heals slowly. In the course of time the condition may involve the glands above the claricle and those in the posterior cervical triangle. Occasionally the axillary glands enlarge, soften and suppurate. The temperature is seldom high, even when the glands break down. The slow course of the disease and a positive tuberculin test are the most important aids in diagnosis.

Treatment. (See page 603.)—As the course is more or less protracted, nothing should be left undone to improve the general condition of the patient. Fresh air, sunshine, and a diet low in carbohydrate are very important. A sojourn of several months or a year to the mountains or seashore is often beneficial. A course of tuberculin injections may be beneficial ("Tuberculin Therapy," page 606). *X*-ray therapy may be of value early, but when softening has occurred surgical interference is indicated. Chronic fistulas often respond to natural or artificial heliotherapy or to *x*-ray. Recurrences are not infrequent, and the cicatricial area is often keloid in nature.

Tuberculosis of the Mesenteric Glands (Tabes Mesenterica).—The mesenteric and retroperitoneal glands enlarge and frequently caseate as the result of a primary lesion in the intestine or from the passage of bacilli through the intact intestinal wall. Park and Krumwiede[61] found about 50 per cent. of abdominal tuberculosis to be due to the bovine type of bacillus.

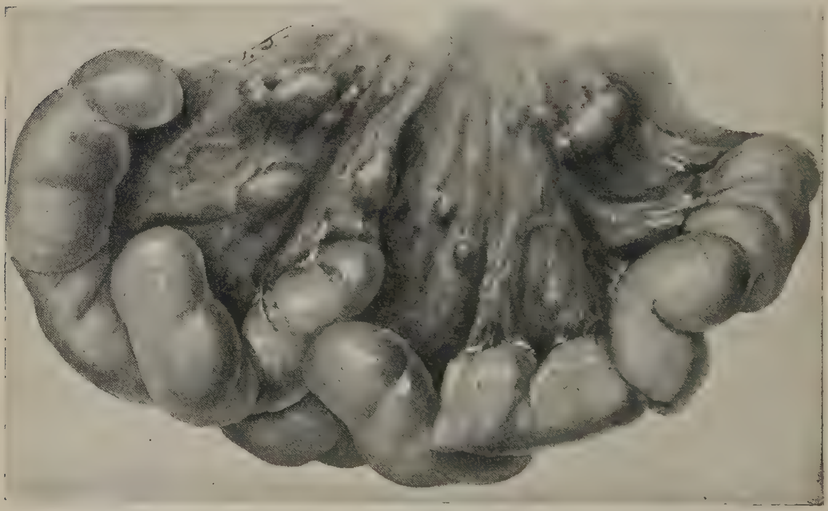


FIG. 343.—Tuberculosis of the mesenteric glands. (The infant died of a miliary tuberculosis of the lungs at the age of four months.)

The diseased glands vary greatly in size from those only slightly enlarged containing tubercles, to those forming larger tumors of fused, caseated masses (Fig. 343). In advanced cases there is more or less involvement of the peritoneum in the vicinity of the glands.

Irregular fever, anemia, loss of weight, anorexia and slight abdominal pain are the predominating symptoms. The general symptoms are frequently obscured by those caused by tuberculous peritonitis or intestinal ulcerations.

Bimanual or deep palpation may reveal enlarged mesenteric glands close to the abdominal wall or against the spinal column. The tuberculin test is almost invariably positive; in case of doubt, the test should be repeated with both human and bovine tuberculin. Chronic appendicitis, intestinal obstruction, and other abdominal conditions must be excluded.

Treatment.—The general treatment outlined under cervical gland tuberculosis is also of value in mesenteric gland disease. Diathermy and heliotherapy are of particular value.

TUBERCULOSIS OF THE SEROUS MEMBRANES

The tubercle bacillus can reach most of the serous membranes. The resulting inflammation produces either a thin serous, or a thick fibrinous exudate.

General Tuberculosis of the Serous Membranes (Polyorrhomenitis).—This rather rare form of tuberculosis occurs mostly in infancy and early childhood. The serous membranes, especially the meninges, peritoneum, pleuræ and pericardium, are involved, either simultaneously or consecutively. It may occur in the course of a generalized tuberculosis or be limited to one or more of the serous membranes. It is probably always due to a tuberculous focus elsewhere in the body. At necropsy the lymph glands in various parts of the body are often found tuberculous. The course is usually acute, or when chronic, death may be preceded by meningitis or an intercurrent pneumonia. The diagnostic tuberculin reaction is always positive. Heliotherapy, x-ray, proper food and fresh air are the main therapeutic measures.

Tuberculosis of the Pleura.—Tuberculous involvement of the pleuræ is often secondary to tuberculosis of the lungs or bronchial glands. In fact, the visceral pleura is rarely normal in any case of pulmonary tuberculosis. There may be a moderate thickening over the diseased area of the lung or firm, fibrous adhesions may bind the lung to the thoracic wall. If the pulmonary involvement is extensive, areas of caseation or pus pockets may be found between the visceral and parietal pleuræ. These forms of pleuritic involvement are, however, of little clinical importance, as the condition is usually overshadowed by the pulmonary condition.

Tuberculous Pleurisy with Effusion.—This clinical type of pleurisy is not common in children and is rare before the seventh year.

Etiology.—The condition is usually secondary to a tuberculous focus in the lungs or bronchial glands. In rare instances it results from extension of a tuberculous process in the vertebræ or ribs. In exceptional instances a pleural invasion by way of the lymph channels from the peritoneum or cervical glands occurs. Trauma of the chest wall is, at times, supposed to produce a "locus minoris resistentiae" whereby a quiescent focus in the chest is re-activated and a pleurisy with effusion results.

Pathology.—The exudate is serous, fibrinous, or purulent. At times, a serous fluid becomes purulent. Small lymphocytes usually predominate if the exudate is serous. The pleuritic fluid is usually found sterile by the ordinary cultural and staining methods, but guinea-pig inoculation of the centrifuged sediment will often produce tuberculous lesions within a few weeks. Scattered tubercles stud the visceral and parietal pleuræ or thick fibrous adhesions may bind the lungs to the thoracic wall.

Symptoms.—If the onset is acute, pain may be present in the involved side of the chest. A slight, irregular fever, cough, anemia and anorexia are usually the only other symptoms. As the disease advances, dyspnea, cyanosis and rapid pulse are present.

Physical Signs.—Fluid in the chest may be the first sign of tuberculous disease. Inspection may reveal diminished movement and bulging of the intercostal spaces of the affected side. If the effusion is on the left the apex beat may be displaced some distance within the mamillary line; if on the right, the beat may be in the axillary line. Palpation usually gives an absent or diminished vocal fremitus; percussion reveals dulness or flatness over the lower part of the chest if the fluid is small in amount, or over the entire side of the chest if extensive. In the former case the line of flatness varies with the patient's position. Distant bronchial breathing may be heard over the area. Friction sounds may be heard some distance above the line of fluid, especially when the process is subsiding. Râles may then also be more audible.

Diagnosis.—A flat percussion note, especially if it is over an entire side of the chest without a history of pneumonia, is very suspicious. Distant bronchial breathing, displacement of the heart, a positive tuberculin reaction, and obtaining a serous exudate on exploratory thoracocentesis are valuable diagnostic aids. A predominance of lymphocytes in the fluid is very suspicious evidence. Producing tuberculous lesions in the guinea pig by injection of pleuritic exudate makes the diagnosis certain. In pneumonia the temperature and leucocyte count are higher. The x-ray will exclude pericarditis with effusion. Fluoroscopic examination will differentiate between old pleuritic adhesions and an active pleurisy.

Prognosis.—The condition may terminate with slow, spontaneous absorption if the process in the lung is not very extensive. Most effusions subside in the course of weeks or months. Old pleuritic adhesions often leave shadows long after the process has healed.

Treatment.—The early treatment should be expectant and symptomatic. Fresh air and sunshine are important. Absolute rest in bed in a semi-sitting position will avoid embarrassment of heart and lungs.

Sedatives, heat, counterirritation or strapping the side with adhesive plaster may be resorted to if the pain is severe.

Should the respiration and circulation become seriously embarrassed by the rapid accumulation of fluid, or if there is no tendency to resorption after a number of weeks, harm may result if some of the fluid is not removed. In fact, the removal of some of the fluid usually hastens recovery. The Potain apparatus is very well adapted for this purpose. Complete removal of the exudate is considered unwise, as the sudden activation of the lung thus produced has caused a apids dissemination of the tuberculous process into other parts of the brod. Aspiration may be repeated if occasion demands. Pneumothoray must be avoided, as it usually masks the subsequent course of the disease.

Tuberculosis of the Peritoneum.—Tuberculous peritonitix is not a frequent form of clinical tuberculosis in this country today, but most cases of chronic peritonitis in children are tuberculous. The condition may develop as a part of a miliary tuberculosis, as a complication of some other form of clinical tuberculosis, or as the sole manifestation of tuberculosis. It is most rare in children between one and five years of age. The incidence of this type of tuberculosis has shown a marked decline since the commercial pasteurization of milk.

Etiology.—The bovine type of bacillus was found to be the etiological factor in about half of Park and Krumwiede's[61] cases. The condition is usually secondary to some small, often distant tuberculous lesion. The bacilli reach the peritoneum through the blood stream as in miliary tuberculosis, by way of the lymphatics, or from caseous mesenteric or retroperitoneal glands.

Forms of Tuberculous Peritonitis.—For practical reasons the principle varieties of tuberculous peritonitis will be taken up under the fibrous, by far the most prevalent, and the ascitic, although intermediate forms occur.

The Fibrous Form.—*Pathology.*—If the examination is made shortly after the peritoneal dissemination, as in some cases of miliary tuberculosis, a multitude of small tubercles may be found on the parietal and visceral peritoneum. In advanced cases flakes of fibrinous exudate adhere, and more or less cover the thickened and congested serosa of viscera and abdominal wall. Large caseous masses are found in the omentum and between the coils of the firmly matted intestines. The latter may be more or less firmly cemented together into a solid mass. Fluid, if present, is either free in the general peritoneal cavity or sacculated by firm, encapsulating fibrous adhesions. The exudate may be serous, seropurulent, or purulent. In advanced stages the intestinal and abdominal walls are often infiltrated and purulent fluid may discharge through fistulous openings in the umbilical region. In some instances fecal fistulas may be present.

Symptoms.—The development is usually insidious and the course chronic. At first there may be no or only slight fever and constipation frequently alternates with diarrhea. The general health may remain fairly good for some time, even after the abdomen has increased considerably in size. Abdominal pain and a feeling of fullness are frequently more or less constant.

Physical Signs.—Abdominal distention is at first due to tympanites and constipation, but as the disease progresses exudate may develop. The latter may disappear spontaneouly after some weeks with a resulting decrease in abdominal circumference. Irregular, nodular masses are usually felt in the navel and cecal regions. The thickened omentum in the epigastrium is usually easily palpated. In spite of the prominence of the abdomen, fluid may escape detection, as the dulness may not shift and "undulation" may be absent. Areas of dulness and tympany may proximate one another. If the pressure is extreme the veins of the abdominal wall become prominent. Albuminuria and edema of the legs and genitals are also frequent manifestations of venous stasis.

Diagnosis.—Abdominal pain, diarrhea alternating with constipation, slight but constant fever, an enlarged abdomen containing nodular masses or sacculated fluid, pouting of the navel with redness or induration in a child with a positive tuberculin reaction makes the diagnosis of tuberculous peritonitis certain. Fecal masses, chronic appendicitis, hydatid disease and megacolon can usually be easily excluded. As the danger of intestinal puncture is too great, abdominal paracentesis as a diagnostic aid should not be resorted to.

Prognosis.—The younger the child the more rapid the course and the more serious the prognosis. Older children occasionally recover spontaneously and completely, but the course is long and exacerbations are frequent. Death may result from obstruction or other tuberculous disease.

Treatment.—Prolonged rest in the recumbent position, heliotherapy, fresh air and a careful diet should be instituted early and continued for many months. The abdomen or entire body should be exposed to the direct rays of the sun or to the mercury quartz lamp radiation (75 cm. distant). Exposure of the abdomen and back should be gradually increased from 3 to 60 minutes. Cases complicated by intestinal tuberculosis are seldom benefited. On cloudy days and during the winter this treatment may be given with the mercury quartz lamp.

The results of laparotomy are discouraging. Adhesive bands causing severe abdominal pain, intestinal obstruction, and extreme ascites are, however, indications for surgical interference, but fecal fistulæ frequently result.

The Ascitic Form.—This type of tuberculous peritonitis is much less frequent than the one just described.

Pathology.—The serous or sero-fibrinous exudate which characterizes this form is the result of an inflammation caused by the tubercle bacillus. The fluid is usually yellow or brown and may be clear, turbid or, at times, somewhat purulent. In the acute stage of the disease free fluid is more or less abundant in the peritoneal cavity; in the subacute, it is often sacculated. Small gray disseminated tubercles cover the visceral and parietal peritoneum.

Symptoms.—The onset may be sudden but is usually gradual. The early symptoms are vague. Fever may be absent during the first few weeks, emaciation, if present, is seldom extreme. Vomiting, colicky abdominal pain and tenderness occur from time to time. After some weeks the abdomen becomes more or less prominent, the distention being due to ascites and tympanites. Anorexia, and constipation alternating with diarrhea are frequently present.

Physical Signs.—The abdominal dulness is often of the shifting type and "undulation" can frequently be elicited. This may be the only sign present. Deep or bimanual palpation may reveal irregular masses of matted intestines, omentum or enlarged mesenteric glands. There may be pouting of the navel.

Diagnosis.—Chronic colicky pains, an enlarged abdomen containing fluid, and palpable masses in a young child with slight fever and a positive tuberculin test make the diagnosis certain. If the first tuberculin test is negative, it should be repeated and bovine tuberculin should also be used.

The conditions mentioned in the differential diagnosis of the fibrinous form can usually be excluded without difficulty. Abdominal paracentesis as a diagnostic aid is contra-indicated.

Prognosis.—In many cases the fluid is slowly resorbed, but not infrequently it returns or the disease assumes the fibrous form. In some instances emaciation progresses and the course terminates fatally in 6 to 12 months; in others, pulmonary or generalized tuberculosis hastens the end. A case may be considered cured four or five years after an apparent recovery without an exacerbation.

Treatment.—The general treatment outlined under the other form is also applicable here. The food should be semi-solid or liquid and given in five small meals. 0.3 gm. (5 gr.) of guaiacol carbonate may be given in milk three times a day. Laparotomy is of little aid and the abdominal incision heals slowly. Heliotherapy usually yields better results than does surgery.

Tuberculosis of the Pericardium.—Tuberculous involvement of the pericardium is even more rare than that of the pleuræ and peritoneum. Scattered tubercles are, however, not infrequently found in cases of miliary tuberculosis. As a rule the condition results from contact of the pericardium with caseous bronchial glands, or a tuberculous pleura.

Pathology.—The most frequent form is that in which the pericardial sac is more or less firmly adherent to the heart. In acute cases there may be sero-fibrinous, hemorrhagic or purulent exudate.

Symptoms and Physical Signs.—Precordial pain, dyspnea and fever are often the only constant symptoms. Signs of cardiac insufficiency may be caused by dilatation and hypertrophy, the result of chronic adhesions between the heart and sac. Effusion is rare. The liver may be palpable and there may be increased cardiac dulness or serous effusion may be detected in the pleural and peritoneal cavities.

Diagnosis.—The condition may simulate that of chronic pericarditis due to any other cause. The *x*-ray is of little aid. A positive tuberculin reaction and other signs of tuberculosis, especially tuberculous pleurisy, may be significant. Guinea-pig inoculation of aspirated fluid may make the diagnosis certain.

Prognosis.—The outcome is usually dependent on the general course of the disease in other organs. Effusions are sledom resorbed spontaneously and fibrous adhesions are usually permanent. The condition may be latent throughout life.

Treatment.—Rest in bed, fresh air, sunlight, proper food and symptomatic treatment may, in time, bring about marked improvement.

TUBERCULOSIS OF THE GENITO-URINARY SYSTEM AND SKIN

Tuberculosis of the Genito-urinary System

Tuberculosis of the genito-urinary tract is very rare in children. A primary lesion on the penis with secondary inguinal gland involvement has been repeatedly observed following ritual circumcision (see "Tuberculosis of the Skin"). A general tuberculosis usually results. Tuberculosis of the testicle, epididymis and female genital organs is extremely rare.

Tuberculosis of the Kidney. —Tuberculosis of the kidney is the most frequent form of genito-urinary involvement. This organ usually becomes infected from a distant focus by way of the blood stream. Ascending tuberculous involvement is extremely infrequent.

Pathology.—In fatal pulmonary tuberculosis it is not uncommon to find a few, small, scattered tubercles in the kidneys. In general tuberculosis miliary tubercles are usually present on the surface and in the substance of the kidney. The infection usually begins in the cortex and spreads to the mucous membranes of the pelvis, calices and pyramids. As a rule, only one kidney is involved. When the tubercles in the pyramids caseate, coalesce and soften, an abscess forms. After some months the destruction may extend to the cortex. When the condition spreads to the tissues surrounding the kidney a deep abscess involving the lumbar region of the affected side results. This is known as a tuberculous perinephritic abscess. In the course of the disease the other kidney may become involved.

Symptoms and Physical Signs.—The clinical picture is like that in the adult except for the addition of a frequent symptom—incontinence of urine. Pain and tenderness in the kidney region of the affected side are absent in the early stage. Hematuria and pyuria may be periodic or more or less constant.

Diagnosis.—Obstinate pyuria, not due to the colon bacillus or cocci, is always suspicious. The presence of blood in the urine over long periods of time may be significant. Finding tubercle bacilli in the urinary sediment or producing tubercles in guinea pigs with the latter makes the diagnosis certain. The tuberculin test is always positive. Ureteral catheterization and functional tests should be performed to determine whether one or both kidneys are involved and to what extent the disease has progressed.

Prognosis.—The course is usually chronic and not infrequently depends upon the outcome of tuberculosis elsewhere. Non-surgical treatment may be successful if the diagnosis is made early. If only one kidney is involved surgery may produce a cure within a relatively short time.

Treatment.—Rest, air, a careful diet, diathermy and heliotherapy may be tried. If the functional tests show no improvement after some time, nephrectomy should be performed. Waiting too long before nephrectomy may endanger the well kidney. If both kidneys are involved, but one is still functioning fairly well, nephrectomy of the other may possibly effect a cure.

TUBERCULOSIS OF THE SKIN

The various forms of tuberculosis of the skin occurring in children are taken up in detail in another volume of this work.

Primary Tuberculosis of the Skin.—The intact skin is invulnerable to the tubercle bacillus. The cases of primary tuberculosis reported in the literature show conclusively that tuberculous disease, especially miliary tuberculosis, may follow a primary tuberculous infection through an injury of the integument. Holt[39] collected 42 cases of primary tuberculosis of the penis following ritual circumcision. The source of infection was invariably an adult with bacillary sputum. Bacilli were frequently found in ulcers of the lacerated prepuce and in the enormously enlarged inguinal glands. Death from a miliary tuberculosis usually followed within a few months after the primary infection. Early excision and *x*-ray therapy of the ulcers and inguinal glands offer the best chances for recovery.

A number of other primary dermatogenous infections with the tubercle bacillus through injury of the skin have been reported. Chancellor[13] describes a case in which a tuberculous lesion appeared on the cheek of an infant following a bite by a tuberculous nurse. Epstein[43] observed a primary lesion on a child's ear following the piercing of the lobe with a needle by a tuberculous mother; and H. Koch[43] tells of the presence of a tuberculous ulcer in an eczematous area on the cheek of a four and half months old infant. In all of these instances the regional lymph glands enlarged and suppurated, and other signs of tuberculous disease subsequently developed in distant parts of the body.

Tuberculides.—These minute lesions of the skin in tuberculous children are frequently of considerable diagnostic value. The slightly raised, red papule is about the size of a typhoid rose-spot (1 to 3 mm. in diameter). It is rather flat and topped by a tiny vesicle, at times surrounded by a narrow, congested zone. When the crust which forms is removed, a round depression remains which usually presents a dry, white, glistening surface with a narrow pigmented border. On the ventral surface of the delicate crust a small projection can usually be seen which coincides with the round depression in the skin. The white scar heals quickly and a lesion runs its entire course in two to three weeks. A crop seldom consists of more than a dozen lesions. The sites of predilection are the arms, lower part of the back and extensor surfaces of the lower extremities. Tubercle bacilli have been found in the lesions. Tuberculides, like phlyctenules, may be the only manifestation of tuberculosis. Some clinicians consider their occurrence of grave significance, but numerous cases have been reported where recovery occurred.

IMMUNITY AND PROPHYLAXIS

Immunity

The introduction of tuberculosis into regions hitherto free from the disease or the infection of emigrants from such places invariably produces a very high incidence of a rapidly fatal form. On the other hand, tuberculosis in countries in which the disease has prevailed for centuries shows a low and rapidly decreasing death-rate. Römer[74] says: "The less tuberculosis in a race the higher the lethality; the more prevalent the disease, the lower the lethality." In Europe, for example, the death-rate from this disease showed a decline before the discovery of the bacillus by Koch. Experimental observations lead us to conclude that immunity is not inherited, but that the offspring of a tuberculous race have a greater power of producing immunity than do those of races recently exposed to the disease.

Immunity is a protective reaction of the body-cells to invading microörganisms. Immunity reactions occur when specific toxins and protective substances (immune-bodies) react. The tuberculin reaction is an immunity reaction and shows, when positive, that the bacillus has at some time entered and the body has responded defensively. Hamburger[33] showed that 90 per cent. of the children of Vienna under 12 years of age were infected. Naegeli[57] and others found that most individuals past puberty have tuberculous lesions at necropsy. Comparing the vast number of individuals who show tuberculous infection with the few who succumb to the disease, we are compelled to conclude that, in most instances, the defensive response of the body-cells—the immunity reaction of the organism—is usually successful.

All statistics show that the lethality is extremely high during the first two years of life and that it decreases with advancing age, in spite of the increase in the incidence of infection. In other words, the power to produce immunity is insufficient or wanting in most infants and young children, a condition analogous to that of adults of non-tuberculous races.

Defense, if it occurs at all, begins at the time of the primary infection and its ability to over-balance the invasion determines whether the patient recovers completely, succumbs in childhood, or later in life. The variable course of tuberculosis depends upon the virulence and number of the invading bacilli and the immunity powers of the affected child. The necropsy findings of Naegeli and others show that a healed focus can produce sufficient protection against subsequent infection.

Héderer[37] has recently tabulated the immunity response as follows:

(*A*) Tubercle bacilli on virgin soil.
Three possibilities exist:
1. If the primary infection is greater than the immunity response of the virgin soil, acute tuberculosis as in infancy and recently exposed races results.
2. If the primary infection is less than the immunity response, quick and complete healing results.
3. If the primary infection equals the immunity response, the immunity increases and there may be latency or progression.

(*B*) Tubercle bacilli on sensitized soil.
Three possibilities exist:
1. If the exogenous or endogenous superinfection is greater than the immunity response, a fulminating type of tuberculosis results, as tuberculous meningitis, miliary tuberculosis, phthisis.
2. If the exogenous or endogenous superinfection is less than the immunity response, there is a tendency to localization and spontaneous healing.
3. If the exogenous or endogenous superinfection equals the immunity response, an equilibrium between the disease and health results. This is the group which responds well to treatment.

If the virulence is overwhelming, the invasion too massive or continuous, immune-body production fails to keep pace, and the disease spreads. If the immunity just suffices to arrest the disease, the latter may lie dormant until some inhibiting factor interferes with immunity production, when a recrudescence occurs. Among such inhibiting factors may be mentioned intercurrent disease, insufficient food, poor air, lack of sunshine, great physical strain, and superinfection.

Children with subacute or chronic tuberculosis produce as insufficient amount of immune-bodies, and the disease slowly progresses. Tuberculous disease makes rapid progress when immune-bodies are absent, as in infants and in recently exposed races; when the body is suddenly overwhelmed with large numbers of bacilli, as after rupture of a caseous gland into the blood stream; and when the immune-bodies are suddenly used up, as in measles. Infection with the bacillus after the first few years of life usually endows the body with sufficient immunity to protect against further and renewed (exogenous) infection.

PROPHYLAXIS

There is no specific method whereby the body-cells can be aided in their manufacture of protective substances. Infection must be prevented. If it has occurred, its advance must be checked; if the disease is already active, the dissemination of the bacilli must be rendered impossible. The three main sources of infection are tuberculous dairy products, contaminated environment and droplet infection. The bacillus must be destroyed wherever it exists and needless exposure must be prevented.

Prophylaxis against the bovine type of bacillus must be thorough, as this source is not negligible and is easily preventable. Unboiled milk should never be given to infants. Acute, tuberculous udder-disease may develop in the internal between veterinary inspections, and raw certified milk may therefore become the source of infection. All milk and dairy products should be subjected to commercial pasteurization. Children convalescing from any illness, especially from intestinal disturbances, should not have raw milk. Vigilance over commercial pasteurization and conscientious inspection of dairy cows has produced an appreciable reduction in morbidity and mortality with the bovine type of bacillus, but still more remains to be done.

Contaminated environment is a potential source of infection. The infant, especially the newborn, is very susceptible to tuberculous infection which may be rapidly fatal. The newborn should, therefore, not only be removed from the tuberculous mother, but from the tuberculous environment. Von Bernheim's[11] experiment illustrates the danger of a tuberculous environment: Of three tuberculous mothers with twins, one of each pair was taken to the country and artificially fed, while the other three remained at home and received breast milk from wet nurses. The three infants in the country remained well, whereas the three in the tuberculous environment contracted the disease and died in spite of breast-milk feeding.

The main source of danger is droplet infection, having its origin in a tuberculous individual. The most painstaking precautions practiced by an infected adult do not render sufficient protection to an infant. The most important weapon against the dissemination of tuberculosis is isolation of the patient. Children under a year should come in contact with strange children and adults as little as possible. One kiss of an infant by a tuberculous individual may produce a fatal infection. In some families successive deaths of children have occurred, the source of infection having been a parent, grandparent, neighbor or nurse—adults who were unaware that their cough was tuberculous.

If the mother has tuberculosis, the only safe plan is to remove the infant at the time of birth and keep it away at least a year or until the condition in the mother has healed. Among the poor, where tuberculosis is more or less endemic, it is often more expedient to keep the patient in an institution until cured. Prenatal counsel between obstetrician and family makes such isolation relatively easy.

A mother known to have had phthisis but who shows no active signs must be considered a potential source of infection and the sputum should be examined at intervals. If the condition of the mother warrants it, the infant may be allowed to nurse. In the case of a tuberculous father, isolation can be maintained with relative ease. He should not enter the room where the child is, and must maintain the most scrupulous hygiene. Temporary absence of bacilli in the sputum does not insure permanent safety.

In a family of moderate means the tuberculous should be persuaded to go to a sanatorium or the infant or child should be transferred to a prophylactic nursery at least for the first year. Should neither of these prophylactic measures be resorted to, the tuberculous patient should occupy a separate room and exceptional care must at all times be exercised in the disinfection of sputum. The child should never enter the patient's room nor should the patient ever come within a yard of the child.

If infection has already occurred, every measure must be resorted to to increase the child's resistance and to avoid superinfection. The best means at our disposal are the so-called preventoria. Preventorium treatment is especially indicated when a poorly nourished, poorly developed child has a positive tuberculin reaction, but no "open" lesion. The institution should be located some distance outside of the city, preferably at the seashore, in the mountains or on somewhat elevated farming country. A. Hess[38] suggests an affiliation with a nearby agricultural school, where the older children may receive training which will equip them for permanent outdoor occupation. Due to the very sharp rise of active tuberculosis during puberty, the time when such care would be most beneficial is from 10 to 14 years for girls, and from 14 to 17 years for boys. They should live out of doors, get wholesome food and be under medical supervision. After a stay of a year or more, the child may be permitted to return home, provided there is no "open" case in the household.

Public schools, health boards, medical societies, farmer alliances, labor unions and church federations can do much to encourage and expand these valuable measures of child welfare. In France the Grancher work is yielding unparalleled results in this respect.

Every child attending public school should have a health chart on which are recorded: Home conditions, possible sources of infection, biennial weight, height, and hemoglobin, and possibly the result of the cutaneous tuberculin test. Negative tests should be repeated at the close of each school session, at which time the teacher should also be examined. School children, who at one time or another have had active tuberculosis, should be segregated in *open-air* or *open-window schools*, for the disease is more likely to become active if they spend the school hours in poorly ventilated classrooms. The open-window schools are more economical and offer better educational facilities.

PROGNOSIS AND THERAPY

Prognosis

The *age* of the child at the time of infection is a very important factor in prognosis. Congenital or prenatal tuberculosis shows the highest lethality. Infection during the first year of life usually produces severe clinical signs within a short time and the prognosis is bad. Comby[15] says, "Tuberculosis in infants is very grave, but not inevitably fatal. The younger the child the less capable of defense and the greater the tendency to become generalized." H. Koch[44, 45] has computed that a child under four years with clinical tuberculosis is 120 times more apt to develop miliary tuberculosis than is a child 10 or 14 years. As pointed out elsewhere, the virulence of the infection and the opportunity for re-infection are also important factors. The highest mortality results when a newborn infant contracts tuberculosis from its mother.

Hahn[31] found the percentage of recovery among 69 infants with positive tuberculin reactions as follows: 17 per cent. of those under 12 months, 26 per cent. of those between 6 and 16 months, and 39 per cent. of those between 12 and 24 months recovered. This shows that lethality decreases with increasing age at the time of infection. Hamburger[33] found that 80 per cent. of the children infected before they were a year old died, while only 10 per cent. of those infected during the second year died.

The clinical form of the disease is also important. If the infection in the bronchial glands has spread to the lung, the prognosis is more grave than it would be if the disease remained confined to the regional lymph glands. In the secondary stage of the disease the prognosis is not absolutely bad. Many survive, although some with scarred neck or deformed spine.

The location of a lesion is of importance in prognosis. Thus, a cervical gland is less likely to disseminate than one in the bronchial glands.

In the tertiary stage of tuberculosis the extent of the lung involvement at the time treatment is begun, the age and general condtion of the patient and the presence or absence of complications (for example, laryngeal or intestinal involvement), are factors which must receive consideration before an estimate of the outlook can be attempted. It is now well known that a certain number of children with pulmonary tuberculosis, even in the first year of life, recover. Tuberculides, once considered of absolutely grave significance, need not invariably imply a fatal course of the disease.

THERAPY

There is no specific therapy for tuberculosis in any of its forms, and our efforts must be concentrated on increasing the resistance of the infected individual by all the means at out command. Conscientious, intelligent and rational care is a vital factor in treatment. Patience and vigilance are essential. Superinfection by means of contaminated food or surroundings, or a tuberculous adult must be avoided. Cure depends on the amount of immunity which is developed and the success of treatment depends on the degree to which this can be augmented by the various aids at our disposal. Treating the tertiary stage and many of the secondary phases of the disease is often a trying and nearly hopeless task, as the immunity barriers are more or less completely broken down.

We must first attempt to arrest further progress of the disease, as only then can we hope for an increase in immunity or resistance. Even when relatively large areas of vital tissue have been destroyed improvement and cure may result, but scar tissue never functions. Trying to cure an advanced stage of the disease, is often futile for we usually start too late. Therapy of the future must pay more and more attention to the early stages of tuberculosis.

Diet.—The classical experiments of Weigert,[83, 84] in 1907, are of fundamental importance in the nutritional therapy of tuberculous children. His observations on young pigs inoculated with tubercle bacilli show that tuberculous infection spreads rapidly when the diet is chiefly carbohydrate, while those pigs fed a diet rich in fats showed no tuberculous infection or only a limited spread of the disease. On the carbohydrate diet, the tissues showed a high water content, a condition known to be conducive to lowered resistance.

As a prophylactic measure it is, therefore, wise to avoid a rapid accumulation of water in the tissues. Water metabolism plays an important rôle in the progress or arrest of the disease, and a diet which insures the largest deposit of fat in the tissues and the least retention of water is the one most beneficial to the tuberculous child. A proper diet for a tuberculous child need not lead to a rapid gain in weight; in fact, the turgor of the tissues may improve greatly even though the weight remains stationary. Czerny,[18] as advocate of the low carbohydrate diet, remarked in 1913 that he never had a case of tuberculous meningitis or miliary tuberculosis develop in the tuberculous patients of his hospital. Schultz[78] points out that a diet may not only affect the chemistry of the tissues, but also the resistance of the tissues to disease.

On the other hand, emaciation in a tuberculous child without pulmonary involvement is frequently due to an improper diet. These children should receive more calories than theoretically necessary for their age and weight, but overfeeding must be avoided. The diet must be nutritious, but easily digestible. There should be more fat and protein and less carbohydrate than usual. The fat is best given in the form of bacon, unsalted butter and cod-liver oil.

Infants should receive breast milk or cream should be added to the usual milk mixtures. At 10 months, beef juice and scraped beef may be given daily. During the second year the diet should consist of semi-solid food, the amount of milk being limited to a quart a day. During spells of anorexia or diarrhea the amount of fat must be reduced. The food should always be prepared and served with ingenuity, and there should be daily variation. Likes should be included if compatible with the prescribed plan. If anorexia occurs, then coercion is necessary.

Menu for a Tuberculous Child of School Age

7 a.m. Cereal cooked in 8 oz. or more of rick milk with butter added, or 8 oz. of cocoa or rich, boiled milk with bread, toast, or rolls and butter.
10 a.m. Sandwiches with chopped egg, cheese, meat or fish.
1 p.m. If soup is desired, grated cheese, cream or butter should be added; potato purée with gravy or butter; finely cut meat and small amount of dessert.
4 p.m. Eight ounces of boiled milk flavored with cocoa or coffee, and three or four pieces of zwieback or 8 oz. junket with crackers.
7 p.m. As 1 p.m., except for the addition of a vegetable.
8:30 p.m. A cup of boiled top milk.

Rest.—All acute cases should have sufficient rest. Where there is a morning elevation of temperature, the patient must be confined to bed, not infrequently for many months. Rest allows an hyperemia of the focus to occur. As fresh, moving air is, *per se*, of great value, the bed should be out of doors whenever the weather permits. Roof, sleeping porch or a large, well-ventilated sunny room with the windows constantly open will be found the best places for treatment. Patients confined to hospital beds in the city are greatly handicapped.

If an active child with a positive tuberculin reaction fails to thrive for some time, more rest should be provided for. Fatigue resulting from long hours at school, long walks, or strenuous exercise, must be avoided. There should be only a half-day session at school, a midday rest period, and breakfast and supper should be eaten in bed. If circumstances permit, a few months of rest should be spent in the mountains, at the seashore or in the pine woods. Among the more moderately situated, the child should be given rest and sun treatment all summer at home. If phlyctenules, pleurisy or hilus involvement are present, even greater restrictions should be made.

If improvement fails to occur with the above amount of rest, if fever persists and subsequent examination shows an advance of the disease, then absolute rest in bed is indicated. Artificial pneumothorax may possibly augment the desired immobilization of an affected lung. Absolute rest in bed should be resorted to only when the condition warrants, as the aeration of the lung, appetite, and the psychic state of the patient not infrequently suffer thereby.

Climate.—That tuberculosis is not a climatic problem is proven by the fact that in the Tyrol it shows a high and increasing mortality rate, while in the foggy British Isles the mortality is low and decreasing. A change of abode is not absolutely necessary if the home conditions are satisfactory. Tuberculous children should be removed from the dust-laden atmosphere of the city. Fresh, moving air is a very essential therapeutic agent in tuberculosis. In a mild climate with good meteorologic conditions, and pure, dust-free air, secondary catarrhal complications are not likely to occur. Ideal conditions are usually met with at moderate elevations (2000 ft.). Mountain climate produces a strong vasomotor and respiratory stimulation which is reflected by an increased metabolism. The patient's room should have a southern exposure, should be dry and well ventilated. Walking and other exercise of ambulant patients must be supervised and graduated to prevent harmful strain.

The *seashore* offers an ideal and convenient opportunity for hardening against sun and wind. The best season of the year is from June to September. A change of climate does not imply that detail of treatment may be neglected. The benefits of the seaside are derived at the beach, not in hotels beyond the dune-line. The remarkable qualities of sea air extend only a short distance beyond the water's edge. At first, only two hours twice a day should be spent there. As the child becomes accustomed, the periods may be lengthened. A cap covering head and ears should be worn the first week. Sun baths at the beach must also be graduated. Sea bathing should not be attempted until the child shows definite improvement. In the beginning, the child should remain in the water only one to five minutes, later on five to ten minutes. The body should feel warm when the child comes out of the water.

Heliotherapy.—Heliotherapy consists in graduated exposure of the body to the direct rays of the sun for increasing periods daily until the resistance so acquired permits a long exposure to light and air. The treatment is usually well borne by children. During the treatment the head should be protected and the room be comfortable for the patient. The child may wear stockings and a gown and should lie on a blanket in the sun. During cold weather, the first sun baths should be given with the window closed, then open, then on a porch and finally out of doors. The gown is raised, the entire legs are covered and kept warm. The back and abdomen are each exposed daily, at first for five minutes, the length of time is increased by one or two minutes each day until an hour or more of direct exposure is reached. The patient must never be allowed to become too cold or too hot. A nurse should be on duty during the entire time of exposure. Rollier[75] of Leysin and others get excellent results with heliotherapy in gland, bone and joint tuberculosis. Only incipient cases of pulmonary tuberculosis without fever may be so treated. The quartz ultra-violet light is of value in the treatment of local tuberculous processes, especially those of the bones, abdomen, glands and skin.

Drugs.—A "therapia sterilisans magna" for tuberculosis has not yet been found. The striking influence of arsphenamine (salvarsan) on the Treponema pallidum has led to a vast amount of chemotherapeutic research with the tubercle bacillus. The chemicals mostly experimented with have been dyes of the methylene blue series and organic compounds of arsenic, copper, gold and mercury. None of the many substances thus far studied acts as a specific.

Cod-liver oil is a valuable therapeutic agent when given in large doses in afebrile tuberculosis, provided the appetite and digestion are good and the bowels not loose. It should be given in small doses at first and the quantity increased to at least 15 c.c. (½ oz.) daily. It may be disguised by the addition of small quantities of a volatile oil or malt, or may be given in orange juice, milk, or coffee. A pinch of salt on the tongue before taking the oil makes it less repulsive. A small dose of the pure oil is as valuable as a large dose of an emulsion which contains but little of the oil.

Creosote, guaiacol and their compounds are of questionable value. If large doses are taken, gastric and intestinal disturbances frequently result. Five drops of creosote carbonate taken in sweetened milk or in capsules two or three times a day may be valuable in anorexia. A favorite prescription is:

Creosoti.................................0.5 gtts. vii
Ol. Morrhuæ.............................100.0 ℥ iii
M. S. Dessertspoonful b.i.d.p.c.

Iodides, especially the iodide of iron, should not be used indiscriminately in tuberculosis, as they seem to stimulate the tubercles, and, tuberculin-like, may cause the lighting up of a quiescent process or a serious exacerbation of a slow process. Tuberculous glands may thus be harmfully over-stimulated. Most authorities agree that iodin is inert. Arsenic and iron may be of value in anemia.

Tuberculin Therapy.—Tuberculin injected into the non-tuberculous fails to produce any immunity against a subsequent infection with the bacillus, nor does it increase the resistance of the tuberculous. It exerts its effect merely through its activation of the focal lesion, whereby healing is possibly hastened.

No unanimity exists as to the value of tuberculin as a therapeutic agent. According to some, it is not only inert, but may be dangerous, as hidden foci may thus be activated. Most authorities agree that:

1. Tuberculin is contra-indicated in all advanced cases of tuberculosis, such as phthisis, miliary tuberculosis, tuberculous meningitis, pneumonia and tuberculosis of the kidney.
2. There is no usable, schematic rule of tuberculin therapy.
3. The patient must be under continuous clinical control.

4. The first injection should be infinitesimal.
5. The treatment should be begun early in the disease.
6. Afebrile cervical gland, skin and bone tuberculosis apparently give the best response.

Czerny[19] points out that for every reported cure by the aid of tuberculin therapy, a similar cure can be shown without its use. He ironically remarks that the mild cases of tuberculosis heal in spite of tuberculin. Rollier[75] does not use tuberculin treatment, and his results are unparalleled. No disciple of tuberculin therapy has discontinued the use of other known therapeutic measures.

Enough tuberculin should be given to affect the local process, but a general systemic reaction—fever, maliase, etc.—should be avoided. Of the score or more methods which have been suggested, that of Cronquist[17] is probably the safest. His initial hypodermic injection in tuberculous children is usually 0.00001 to 0.00002 gm. of Old Tuberculin in sterile normal salt solution. In those who are likely to react, for example, cases of marked glandular involvement, he begins with 0.000002 or even 0.0000002 gm. (1 gm. of Old Tubercuin is the equivalent of 1 c.c. A 1:10,000 solution is made from the 1:100 solution by diluting 0.1 c.c. of the latter with 9.9 c.c. of sterile salt solution.) He gives two injections a week, the dose being increased as follows: 2, 3, 4, 6, 8, 10, 12, 15, 20, etc., until 0.05 or 0.1 gm. is reached. The maximum dose is then maintained for weeks or months. Systemic reactions must be avoided, and at the end of the treatment the cutaneous tuberculin test is usually found negative. He interprets a permanent negative reaction as proof that a cure has been effected.

Müller[56] divides a dose of tuberculin by injecting simultaneously a fraction of the dose in various parts of the body. H. Koch[46] dilutes the tuberculin in 20 to 30 c.c. of normal salt solution, whereby systemic reactions are less likely to occur. He begins with 0.00001 gm. and, as a rule, reaches 0.001 gm. in eight weeks. He gives a total of eighteen injections about four days apart.

Petruschky[66] finds the percutaneous administration of tuberculin simple, inexpensive and effective. The patient rubs a definite and increasing amount of a tuberculin ointment into the skin every three days. Numerous clinicians claim to have obtained excellent results with this method in gland, bone and joint tuberculosis. Von Hayek[36] and others favor this kind of therapy in the treatment of the primary and secondary stages of the disease. They hope in this way to influence beneficially the specific resistance of the body at a time when the future course of the infection is determined.

Some clinicians, especially those in continental Europe, consider Deny's bouillon filtre (B.F.), tuberculin residue (T.R.), and bacillary emulsion (B.E.), more satisfactory than Old Tuberculin, as these preparations are standardized. The safest Old Tuberculin is that prepared according to Rosenbach.

Other Specific Therapy.—Many sera and vaccines have been recommended, but all have been found to be of little or no value. Attention is again focused on the therapeutic use of Friedmann's turtle tubercle bacilli. Klopstock[41] reviews the literature on the subject, but much work must still be done before any conclusions can be drawn. The use of any therapeutic measure requires, not only a great deal of experience and skill, but also an unbiased judgment.

Sanatorium Treatment.—Besides offering the best treatment for active tuberculosis, the sanatorium protects the other members of the family against massive and repeated infection. The strict discipline acquired while in the institution is an added protection when the patient returns home. A sanatorium should be equipped to adequately care for incipient and advanced cases. There should be sleeping porches, solaria, operating rooms for pneumothorax and other surgery, *x*-ray laboratory and a good kitchen, all under competent medical supervision. Carefully compiled statistics show that there is a paucity of such institutions, that patients seldom stay long enough and must often wait too long before being admitted. Sanatoria are caring for only a fraction of the active cases of tuberculosis in the United States, and a high mortality rate continues.

Diagnostic acumen must be perfected so that the earliest stages of active disease can be properly treated. We wage a battle against odds when we wrestle with the tertiary stage of tuberculosis. We fail to accomplish the most important task in prophylaxis and therapy until we treat the primary and secondary stages of this dreaded disease.

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CHAPTER CXXIII

HEREDITARY SYPHILIS

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HISTORICAL

While the exact origin of syphilis will apparently be a never-ending subject of historical controversy, the view most generally accepted is that it was a disease endemic in the West Indies and was carried from there to Europe by the early Spanish explorers. Certainly as a specific entity it was not recognized in Europe until after the return of Columbus from America. It was but a few years after this (1498) that Torella mentioned syphilis in the nursing infant. This is again mentioned by Cataneous in 1505 and in 1508 Vella discussed it as a contamination from the nurse. It is to Paracelsus in 1529, however, that the first mention of syphilis by inheritance must be attributed. Although the condition is mentioned by him several times in a vague way, the expression "*—fut morbus hereditarius, et transit a patre a filium*," clearly defines his opinion as to the nature of syphilis in the infant.

Farrier some years later (1553) speaks of transmission during intra-uterine life. Roudelet in 1560 stated he had seen a child stillborn with the pustules of the "morbi gallici," and Ambrose Paré a year later mentioned having seen infants born with the disease. A number of other sixteenth century physicians might be quoted but these instances suffice to show the concept of syphilis in the infant at that time. Little was added to our knowledge in the seventeenth century, but the eighteenth added considerably. The clinical descriptions of Boerheave and of Rosen von Rosenstein in particular are splendid. Later Hunter clouded the subject with his position in regard to the non-infectiousness of hereditary syphilis. In the latter half of the eighteenth century and the early part of the nineteenth the French school in particular was active in the study of the disease and the names of Faquer (1783) Mahon (1804) Bretin (1810) and others belong to this epoch. In connection with this period must be mentioned the special hospital for syphilitic women and their offspring established at Vaugiard in 1780.

The nineteenth century was marked by advances in our knowledge of the clinical manifestations, the pathology, and the modes of transmission of the disease. The names of Diday and Fournier in France, of Hutchinson and Colles in England, of Virchow and of Hochsinger of Austria stand out prominently among the hundreds who contributed in a greater or less degree to our knowledge of hereditary syphilis during this period.

But it was the opening decade of the twentieth century that added more to our knowledge of syphilis, it may be said without exaggeration, than the three previous centuries. In 1903 Metchnikoff and Roux were able to transmit the disease to chimpanzees, thus opening the opportunity for experimental studies. In 1904 Wassermann described the application of the complement fixation test to syphilis—the one most valuable means of diagnosis. In 1905 Schaudinn and Hoffman discovered the causative organism—the treponema pallidum, and in 1910 Ehrlich announced "606" to the medical world, opening the therapeutic field anew. Thus in the space of one decade strides of enormous importance were made in etiology, pathology, diagnosis and treatment which have enabled us to go over the field of hereditary syphilis with a new light.

Much of the old has necessarily been discarded, much has been revised, and much has been cleared of obscurity. It is with these new factors in view and the information they have yielded that the subject must be discussed at present. Without minimizing the value of the painstaking studies of the past it is better to drop them from discussion, as they are now chiefly of historical interest, and in a sense consider the subject as having its origin in this the first part of the twentieth century.

DEFINITIONS

Hereditary or Congenital.—By some the term "congenital syphilis" is preferred to "hereditary syphilis." The argument is that the disease is an infectious process transmitted to the fetus during intra-uterine life and is not a condition transmitted through the sperm cell or ovum, as for example hemophilia or supernumerary digits which are handed down from parent to child through generations. The matter resolves itself into a question of the use of terms—shall "hereditary" be limited to a strict biological sense of cellular transmission, or used in a general sense of something passed from the parent to the child, as for example we quite properly use the word when discussing a position or a title to property. Congenital as it is commonly used in medicine applies to any condition present at birth—not one necessarily involving in any way the question of its presence or possession in the parent being transmitted to the child. Thus a congenital club-foot or a congenital amputation is a condition present at birth but not one transmitted from parent to child. What we wish to stress in the disease under discussion is that it is a condition in the parent which is transmitted by the parent to the child. This is the essential point. Although neither adjective wholly expresses the exact situation it is for the above reason that we prefer the term hereditary or inherited syphilis to the term congenital syphilis. The important thing is the knowledge of what is taking place and not the name by which it is designated.

Stages or Groups.—For many generations it has been the custom to divide acquired syphilis into certain stages. In a similar way it is convenient to divide hereditary syphilis, from a clinical basis, into several stages. In a broad sense there is a certain similarity between the acquired and inherited groupings. We must remember, however, that it is one and the same infection with which we are dealing and that there is little real pathological basis for the classification.

We have found it convenient in our studies to divide hereditary syphilis into four groups:

1. Fetal syphilis.
2. Infantile syphilis.
3. Late or tardy syphilis.
4. Latent syphilis.

In a gross way certain symptoms or groups of lesions are attached to each of these periods but the lines are not absolutely defined. Lesions found in the stillborn infant or the infant at birth, developing during intrauterine life, are designated "fetal." Lesions appearing after birth and during infancy are spoken of as "infantile." In many children a new group of lesions appears during later childhood and these are commonly known as "late" or "tardy." After birth, either before the appearance of infantile lesions or between a group of early and a group of late lesions appearing perhaps after an interval of months or even years, there may be a period in which manifest signs of an infection are lacking. During this time the infection is designated as "latent." The division into these groups is by no means absolute. Not infrequently lesions of the so-called "late" group appear during infancy and we know that certain so-called "specific lesions" of late syphilis, as Hutchinson teeth, which belong to the second dentition and hence are not seen in infancy, are due to interference with growth and development during infancy and are not the result of the syphilitic process becoming active during later childhood. In a certain number of cases so far as can be ascertained the first signs of the luetic infection develop in later childhood. It may be that in some of these cases early signs were very slight and overlooked.

Why such variations occur in the way the disease manifests itself we do not know. While it is obvious that infection of the fetus takes place early in pregnancy in those instances ending in abortion or that without infection of the fetus itself syphilis of the placenta produces early abortion, the truth of the inference that infection takes place late in pregnancy in the cases first showing signs after birth has not been demonstrated. The fact that 37 per cent. of syphilitic infants give a negative Wassermann at birth strongly indicates an infection late in pregnancy. Is the explanation of the cases showing their first signs late in childhood that while in utero a stage has been passed through comparable to that of the infant showing manifest signs? Does the explanation for latency lie in the question of quantitative infection or tissue reaction? These and similar questions have at present no satisfactory explanation and they furnish an important field open for experimental research in the study of hereditary syphilis.

Regardless of the explanation of the pathogenesis, for descriptive purposes the classification of lesions into these four groups is practicable and convenient and will be used without further explanation.

Wassermann Reaction.—It is not within the scope of our subject to enter into a discussion of the Wassermann reaction and its technique, limitations, nature, etc. These are amply discussed in textbooks of serology and a certain amount of knowledge regarding these points is presupposed.

There is no other form of syphilitic infection which gives a higher percentage of positive reactions or in which the reaction is of more value from a standpoint of diagnosis. In studying hundreds of cases of hereditary syphilis in the past 10 years we have found that over 95 per cent. of the untreated syphilitic infants and almost 100 per cent. of the late untreated cases have reacted positively. In one series of over 300 consecutive cases the percentage of positive reactions was about 98.

Positive reactions in non-syphilitic infants are rare—so rare the technique must be questioned. A few very young infants with syphilis fail to react, but if tested after an interval of time the reaction becomes positive. In our more recent investigations 100 per cent. of the syphilitic infants gave a positive reaction after two months.

From a practical standpoint the Wassermann reaction is to be looked upon as a symptom or an indication of the presence of an infection. In the latent group it may be the only indication, or the other symptoms may be so slight that they are ordinarily overlooked. Again and again we have seen these cases with no clinical evidence of syphilis but with a positive Wassermann reaction suddenly develop signs of clinical activity. In such latent cases it is not difficult to provoke active symptoms by instituting vigorous treatment.

The importance of the Wassermann reaction as a guide to treatment is discussed under therapy. Like all delicate laboratory tests the value of the reaction is absolutely dependent upon the technical skill with which it is made and controlled.

Throughout the remainder of the chapter the abbreviation WR will be used for the Wassermann reaction with the symbols + or − meaning a positive or negative reaction respectively.

INCIDENCE OF HEREDITARY SYPHILIS

It is practically impossible to give anywhere near an exact estimate of the frequency of hereditary syphilis. The frequency of infection varies with the occupation, social status, race, nationality and numerous other factors on the part of the parents. Vital statistics are too inexact to throw any light on the situation and no effort to make the disease reportable has been made with any degree of seriousness or promise of success. In the U. S. Registration Area for the years 1910 to 1913 1 per cent. of the infant deaths was reported as due to syphilis and 0.8 per cent. of the total deaths under 10 years. These are minimum figures. Such figures as have been gathered would seem to show that in the United States somewhere around 10 per cent. of the adult male population has syphilis. Jeans by collecting studies made in different cities found that in over 5000 pregnant married women in hospitals, slightly under 10 per cent. were syphilitic. This percentage cannot be applied generally. There are no figures available of a large number of unselected children. Nearly all of the studies of the incidence in children have been made on dispensary or hospital material. Our own hospital material in St. Louis showed 108 cases among 5185 new patients in 1913, or approximately 2 per cent. Of these 854 were infants under one year with a syphilitic incidence of 4.9 per cent. Latent cases are not included in these figures as it was not a complete WR study, and hence the figures are to be regarded as minimum. Jeans and Cooke studied the fetal blood and placenta in 2000 maternity cases in St. Louis. Among negro babies they found 15 per cent. infection, among the infants of the poorer whites 2.0 per cent. and among the infants of the middle and well-to-do classes less than 1 per cent. Austin and Churchhill in a Chicago hospital found an incidence of 3.3 per cent.; Whitney reported 26 positive WR in 890 consecutive admissions to a children's out-patient service in San Francisco. Our own figures show a rate two and one-half times as great among the negro children as among the white.

The Wassermann reaction has shown that many infants and children without clinical evidence of syphilis have a latent infection. Thus 1 per cent. of our apparently non-syphilitic babies give a positive reaction. Wirz has recently reported that 3 per cent. of the apparently non-syphilitic school children in Munich have a positive WR.

It is useless to attempt to estimate the number of syphilitic children. We know that there are many more than we estimated a few years ago, and that in addition to the total number of living children the loss of potential life as a result of syphilis makes the condition one of importance from a social and public health standpoint.

FAMILIAL SYPHILIS

Although as previously stated we possess no accurate figures as to the incidence of herditary syphilis, the influence of syphilis in the parent upon the offspring has been quite thoroughly studied. Starting from the standpoint of the adult, it seems probable from the limited figures available that somewhere between 20 and 30 per cent. of the marriages in which one or the other contracting parties is syphilitic are sterile. This in contrast to the 10 per cent. of sterile marriages from all causes. In the remaining 70 per cent. of syphilitic marriages in which pregnancy takes place it is well known that a large percentage end in either miscarriages or still births. From 15 to 25 per cent. of fetal deaths from all causes are due to syphilis, the variation depending upon the type of material studied.

Further, among the children born alive many die in infancy, and in addition a large percentage of those living bear the marks of a syphilitic infection. The difference between syphilitic and non-syphilitic families is shown in the following summary. The table was obtained by adding the figures of Jeans for 100 syphilitic families and 200 non-syphilitic families in St. Louis and 150 syphilitic families and 150 non-syphilitic families reported by Harmon from London. Both represent families of the same social strata.

Table I

	Number families	Pregnancies	Miscarriages and still births	Infant deaths	Living healthy children	Living syphilitic children
Syphilitic..............	250	1332	303 22.7 %	280 20.8 %	243 18.3 %	580 or
Non-syphilitic..........	350	1712	160 9.7 %	223 13 %	1303 76.0 %	(38 %)

These figures show graphically the tremendous cost of syphilis to the race. Some larger groupings may be quoted in regard to individual points. Thus among a total of 4148 pregnancies in syphilitic families 1258 or 30.3 per cent. terminated in either a miscarriage or a still birth—three times the figures that might be expected to be found in a similar number of unselected families from the same social plane.

Turning now to the living syphilitic births. Either as a direct result of the syphilis or secondarily due to the low resistance of the syphilitic infant to intercurrent infections the death rate in infancy is high. Among 1366 living births in syphilitic families 412, or 30.2 per cent., died in infancy—in contrast to 223 deaths among 1498 non-syphilitics. Or, speaking in terms of infant mortality rate, the death rate for syphilitic infants is 302 as contrasted with 150 for non-syphilitic infants of the same economic conditions. Only a small percentage of the children born of syphilitic families escape infection. Thus of 2450 pregnancies reported by various observers in which the living children were carefully examined (including WR) only 408 or 16.6 per cent. were free from the taint. Among non-syphilitic families the living healthy children amount to about 75 per cent.

To summarize, approximately one-half of the pregnancies in syphilitic families end in death before term or during infancy. Of the one-half with surviving children only a third lead to healthy offspring, and the balance—30 to 40 per cent. of the total—result in children with an hereditary syphilitic infection. And when one examines these mentally and physically defective children with the luetic infection, the cost of syphilis to the community is realized. There is no other disease or condition in which the "sins of the parents," so to speak, take such terrible vengeance upon the innocent blameless child.

TRANSMISSION

For many years considerable controversy has taken place over the question of the manner of transmission of hereditary syphilis, and a large part of the discussion of the subject has centered around this point. We are able today to dismiss most of the discussion without comment, for the discovery of the cause of the disease and of the Wassermann reaction has made it chiefly of historical interest.

For years a group of syphilographers held to the view that syphilis in the father could be transmitted to the child *in utero* without the mother becoming infected at the same time. In support of this view was advanced the fact that while as a rule the father was admittedly syphilitic, many women without a history or signs of lues bear syphilitic children. As further evidence was advanced the clinical observation that at times the specific treatment of the father was followed by the mother bearing normal children after having previously given birth to a syphilitic child. Also that a mother may bear a syphilitic child to a syphilitic father and later a healthy child to a non-syphilitic father. These observations may clearly be circumstance as it is not at all a general rule and furthermore, it is not uncommon for an admittedly syphilitic woman to bear non-syphilitic children after having given birth to one or more who were infected. It is not at all uncommon, however, for a woman without demonstrable lesions or a known infection to give birth to a syphilitic child, and it was these cases which led to the belief that germinal transmission from the father took place. In the instances where the mother was clinically syphilitic, infection through the placenta was generally recognized and accepted as the mode of transmission.

The treponema pallidum is from 6 to 15 microns in length while the head of a spermatazoon is from 3 to 6 microns in length. Unless the treponema undergoes some morphological change in a life cycle, with which we are unfamiliar, the physical impossibility of the spermatazoon carrying the microörganism is apparent. While a number of "granular forms" of the treponema have been described, there is no consistency in the findings nor have these forms been found except by a few observers. The demonstration of such forms would greatly simplify the pathogenesis of the disease could they be shown as actually existing. Further we know of no example of germ-cell transmission of an infectious disease in human or animal pathology. Although it has been shown that the seminal fluid of men with florid syphilis is infectious, and further that the infection may be transmitted by the transplantation of testicles from syphilitic apes, these experiments only prove the infectiousness of the seminal fluid and transplanted glands, and in no way furnish evidence in support of the theory of germinal transmission.

The explanation lies rather in the fact that the "apparently healthy" mothers have syphilis. Perhaps the most valuable purpose of the WR has been to enable us to recognize latent syphilis. In our clinic in St. Louis 74 out of a series of 85 mothers of syphilitic children (87 per cent.) denied any knowledge of being infected with the disease, and excepting a history of repeated abortions and still births, there was nothing in their past medical history to indicate an infection having taken place which might have been overlooked. Yet of these 74 women 67 or 90 per cent. gave a positive WR. In all but one of the negative cases an interval of at least 10 years had elapsed between the birth of the last syphilitic child and the time the test was made. Figures for other clinics substantiate these findings. If, as has been suggested, the mother has simply a passive immunity the WR would not persist. We are justified in concluding from the many studies on this point that syphilis in the child means syphilis in the mother.

A woman may become infected previous to the time of the conception of the child, at the same time, or after conception has taken place. The last two are uncommon and in by far the majority of cases the woman is syphilitic previous to the conception of the syphilitic child. This classification into pre-conceptional, conceptional, and post-conceptional syphilis, so frequently mentioned in the older textbooks, is relatively unimportant.

In this connection must be mentioned the observation of Colles, published in 1837, who noted that the apparently healthy mothers of syphilitic children have an immunity to syphilis, or, more correctly, do not contract syphilis when exposed to infection. This became known as "Colles' law" and as such has been handed down by succeeding generations of physicians. This immunity to infection has had various explanations—the generally accepted one being on the basis of the passage of an immune body through the placenta from the syphilitic child. We know now that the "apparently healthy" mother is "immune" simply because she has the disease in a latent form. The question today is rather the *modus operandi* whereby a woman can be infected by her husband and become capable of transmitting the disease to her offspring without seemingly manifesting any signs of the disease. It must candidly be admitted that we have no satisfactory explanation of this phenomenon.

In 1876 Kassowitz announced that as a general rule the degree of transmissibility of syphilis to the offspring gradually diminishes in proportion to the duration of the disease. That is, that in a given family the first pregnancies end in abortion, then come still births followed in subsequent pregnancies by living syphilitic children, and finally, healthy children may be born. In a general way this rule is correct although not infrequently exceptions occur. Living healthy children and living syphilitic children not infrequently alternate. The starting point may be anywhere in the series. In our material approximately three-fourths of the families follow Kassowitz's rule. As a matter of historical interest it is worthy of note that Hutchinson very clearly set forth this same law in a book published in 1863, 13 years before the published observation of Kassowitz.

Transference to Third Generation.—While the transmission of the syphilitic infection to the third generation is theoretically possible and reports of such cases have been published, it is interesting to note that up until the present time such transmission, when tested in the light of our present knowledge, has not been proven. We have seen several cases in three generations but there has always been the possibility that in the second generation the infection was acquired rather than inherited. In order to prove the transmission we must have a positive WR in the child, its mother and its maternal grandmother, together with evidence (as WR or typical lesions in brothers and sisters) of the infection in the mother (the second generation) being of the hereditary type. The children of known hereditary syphilitics who have so far come under our observation have not had syphilis although not infrequently they have been subnormal.

PATHOLOGY

Etiology.—Syphilis in the infant is caused by the same organism as syphilis in the adult—the Treponema pallidum (Spirochaeta pallida) described by Schaudin in 1905. The organism as shown in dark field illumination or in the tissues by Levaditi's silver impregnation stain averages from 7 to 10 microns in length and has from 8 to 20 spiral turns. The tissues of the syphilitic fetus furnish one of the best means

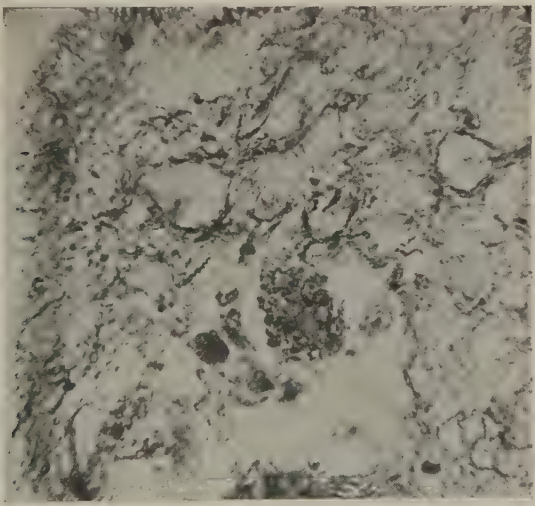

FIG. 344.—Treponema pallidum in tissues. Levaditi's stain.

for demonstrating the organism (Fig. 344). Considerable controversy exists as to whether or not the organism undergoes modifications or changes in a life cycle. A number of granular and other forms have been described, but at the present time their importance and relation is very uncertain. A theoretical acceptance of a life cycle for the organism does much toward clearing up the unclear field of transmission, but unfortunately at the present time the evidence of a life cycle is lacking.

In hereditary syphilis the primary stage of acquired syphilis—the chancre—in which infection takes place through an epithelial covered surface, is lacking. This is a fundamental difference. In hereditary syphilis the infection is intra-uterine and hence by the blood stream through the placenta. The result is a general vascular dissemination of the organism throughout the tissues of the fetus. Theoretically one would then expect the most marked and constant changes in those tissues most intimately a part of fetal circulation with an abundant blood supply, and this is exactly what is found. Tremendous quantities of organisms are found in the blood invading every tissue and in the secretions and excretions. They are found most abundant in the liver and suprarenal bodies. In short, as infection takes place through the blood we are dealing in hereditary syphilis with a septicemia.

Pathological Anatomy.—The fundamental lesion found in hereditary syphilis is an hyperplasia of the connective tissue. Essentially it consists of a periarteritis about the smaller blood-vessels with proliferation of embryonic connective tissue cells. In direct relation to the extent of the process and the degree of connective tissue production various anatomical and structural changes are produced which lead to alterations in and interference with the growth of the various organs and tissues. In itself this typical chronic diffuse interstitial change is not specific, the histological diagnosis is only absolute when the treponema pallidum is found. Hand in hand with this hyperplasia of the connective tissue an hypoplasia of the parenchyma takes place. While it is generally accepted that this is in large part a mechanical process secondary to the overgrowth of connective tissue, it is also possible that there is some direct action of the syphilitic toxin, or reaction of the cell to this toxin, which inhibits the normal development of the parenchyma. Thus at times there are persistent remains of embryonal tissue in the pancreas and other organs and a lack of development of the glomeruli in the kidney, which are attributed by a number of authors to some such direct inhibition.

As pointed out, a general vascular infection takes place and the organisms are found most abundantly in the tissues with the most extensive vascular supply. In fetal life the tissues and organs with abundant vascular supply are the ones with most active function and where most active growth is taking place. Hence as a corollary we find the chief histological changes in hereditary syphilis in those organs which are the site of active growth and function in fetal life. With these general statements the lesions found in individual organs or systems may be described.

Spleen.—Hypertrophy of the spleen is one of the most common lesions found in hereditary syphilis. In nearly 90 per cent. of the syphilitic infants we have seen the spleen has been easily palpable. According to autopsy figures obtained by Parrot and Marfan the spleen of the syphilitic fetus or syphilitic infant in the first month of life is from four to five times that of the weight of the spleen of non-syphilitic newborn infants. This hypertrophy is practically a constant finding with older syphilitic infants.

In its earliest and simplest form the enlargement is due to intense congestion of the splenic pulp, Malpighian follicles and vascular spaces. This is followed and accompanied by interstitial changes resulting from the perivascular proliferation of connective tissue cells. In some instances this interstitial change becomes marked and the spleen may take on an irregular knotty appearance. Small miliary gummata similar to those more frequently found in the liver may occur. In "late" syphilis larger isolated gummata have been described.

Liver.—As a result of its position in the fetal circulation the liver is constantly the seat of syphilitic changes. It is the organ of choice for demonstrating the treponema in the tissues and by Levaditi's method they are found in the lumen of the vessels, outside the vessels, in and about the bile ducts, and even within the liver cells themselves according to Levaditi and others.

The gross and microscopical appearance of the liver varies according to the stage and age of the lesion. In its earliest stage the liver is large and congested. Histologically there is a proliferation of embryonal connective tissue cells but no cellular lesions are present. As the lesion progresses the production of connective tissue takes place, at first peri- and multilobular and eventually intralobular. During these changes the liver remains large but becomes more hard and firm. It takes on a yellowish-brown color which together with the firmness gives it a flinty appearance—the "foie silex" of Gubler. As the connective tissue proliferates and secondarily contracts irregularities develop and the cut surface becomes rough and uneven. The changes may proceed unevenly, different parts of the liver being involved to a different degree with the result that in time marked deformities in the gross appearance of the organ may occur. Histologically firm fibrous tissue is seen forming dense scars at places. The liver cells become atrophied and show degenerative changes to a greater or less degree. The change is usually a fatty degeneration but amyloid change is found at times.

At times when the liver is cut small minute yellowish-white points are found in the cut surface. Histologically these are found to be made up of collections of embryonal cells. They are the so-called "miliary gummata" of Virchow or "miliary syphilomata." Because of their resemblance to grains of semolin scattered over the brownish flinty cut surface Gubler spoke of this as the "foie silex avec grains de semoule" Gummata are also found and may reach considerable size, but they are uncommon. They are found typically in the late or tardy type and resemble the lesion of acquired syphilis.

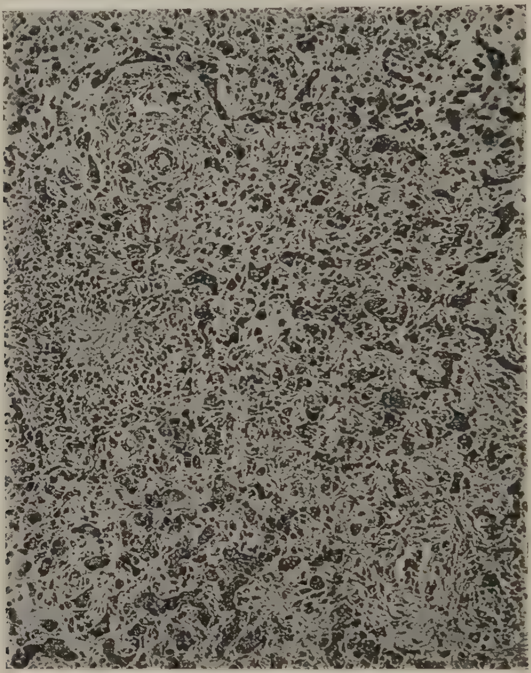

FIG. 345.—Interstitial hepatitis.

Stomach and Intestines.—Lesions of the stomach and intestines are not infrequent but are usually associated with other more severe and prominent visceral lesions. Several types of changes occur—the most common being a diffuse infiltration. The small multiple miliary gummata are also found at times. Infiltration of Peyer's patches occurs and cases of perforation with secondary peritonitis have been described. The meconium of the newborn syphilitic infant frequently contains enormous quantities of the treponema pallidum.

Pancreas.—Lesions of the pancreas are less common. The interstitial overgrowth may be so extensive, however, that it very largely replaces the glandular tissue leaving very prominent islands of Langerhans which are involved to a much lesser degree. Opie has noted the persistence of fetal tissues from inhibition of development.

Adrenals.—Grossly the adrenals of the syphilitic fetus are frequently found very much enlarged. Later the glands may become indurated from the interstitial changes which are largely capsular. As a result of the great vascularity of the adrenal glands treponemata are found in them in abundance.

Kidneys.—Involvement of the kidneys is usually present to some degree. Hecker claims that histological changes can be demonstrated in 90 per cent. of the cases. In addition to a perivascular infiltration which may be so extensive as to produce an actual interstitial nephritis, it is quite usual to find some degree of inhibition or retardation of development of the cortex with the persistence of fetal tubules if the organ is involved to any extent. Small gummata are rarely seen—albumin and casts are sometimes found in the urine of the syphilitic infant and the treponema has occasionally been demonstrated.

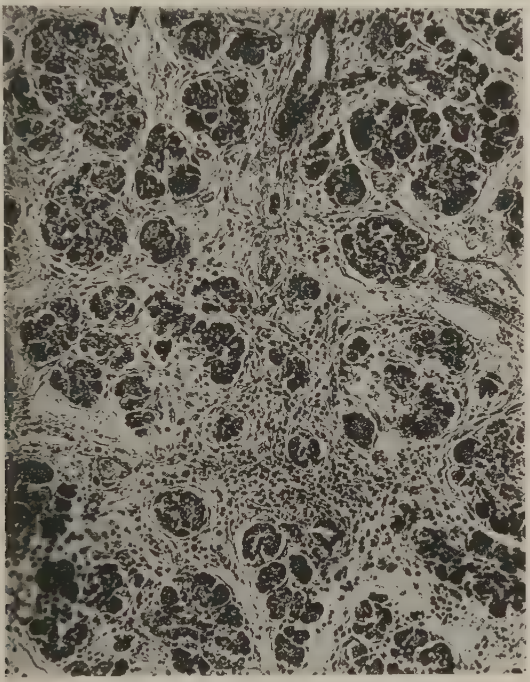

Fig. 346.—Interstitial pancreatitis.

Testicle.—The testicle is not infrequently found enlarged in the syphilitic infant and at times breaks down and suppurates. Later the secondary contraction following the overgrowth of connective tissue may produce a small indurated knotty testicle which is seen at times in older children with hereditary syphilis. The epididymus is rarely involved as the changes are usually limited to the body of the testicle.

Larynx.—The larynx is very frequently the seat of either catarrhal or ulcerative lesions. These may ultimately lead to some degree of stenosis developing in later childhood.

Lungs.—The lung is a frequent seat of syphilitic changes. According to Castens, Heller, and others lesions are found in over 50 per cent. of the cases coming to autopsy. Apparently any degree of involvement is incompatible with life as pulmonary changes are found in the stillborn and early infantile cases and only exceptionally in older children. Several quite distinct processes occur which may be combined in individual cases with the result that marked variations in the gross and histological picture of the lung are seen.

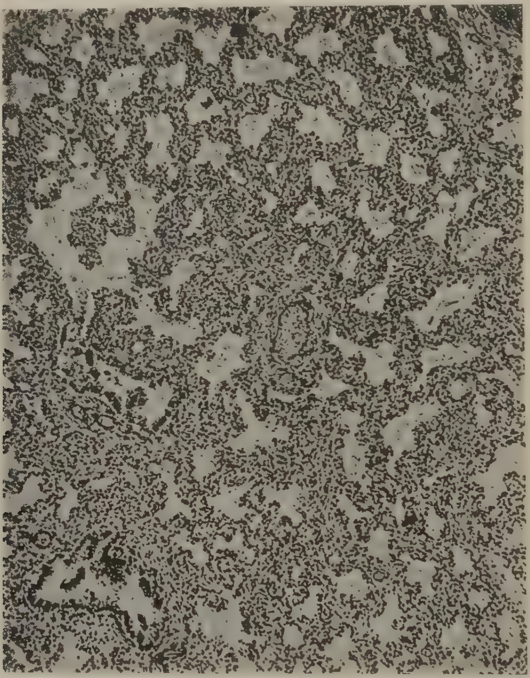

FIG. 347.—Interstitial change in lung of syphilitic infant.

The simplest and most frequent lesion is some degree of interstitial connective tissue hyperplasia. In an early stage this may be chiefly cellular, producing an histological picture somewhat resembling that of a sarcoma. A later stage shows distinct fibrous changes with a tough firm lung having an irregular cut surface. There is usually some congestion accompanying the interstitial changes so that the lung is heavy and voluminous. In the cases dying after birth patches of bronchopneumonia are found at times. The interstitial hypertrophy may be quite uniform in distribution throughout the lung or it may be uneven and in patches.

A second type with which some degree of interstitial change is usually associated is the "pneumonia alba" of Virchow. This is seen almost without exception in the stillborn syphilitic. Grossly areas throughout the lung, or even the entire lung, present a picture somewhat similar to that of the stage of gray hepatization in pneumonia. Microscopically the alveolar spaces are found filled with desquamated epithelial cells of which a large part are embryonal in character. Many of the cells have undergone a fatty degeneration.

In a third type either small miliary gummata are found scattered throughout the lungs, or gummatous areas which reach the size of a cherry may occur.

Thymus.—Similar to the picture in other glands hypertrophy of the thymus is not uncommon. Because of its anatomical situation pressure symptoms from marked syphilitic enlargement have been reported a number of times. In addition to the usual interstitial changes the cyst-like Du Bois abscesses are very frequently seen in the thymus of the syphilitic fetus. Although these cystic structures filled with a purulent material are quite characteristic of inherited syphilis they are not specific for the condition as they are found at times in infants with other conditions.

Blood-vessels and Heart.—As previously noted, the typical change in inherited syphilis is a periarteritis about the smaller blood-vessels and further this change is most commonly noted in the organs with the most marked vascularity. The endarteritis so frequently met with in acquired syphilis on the other hand is only exceptionally encountered in an hereditary infection. Weisner has described an infiltration into the outer layers of the media and adventitia of the aorta and its great branches more or less completely blocking the vasa vasorum, which he considers as quite specific of hereditary lues. Lesions of the heart have been considered as rare by most authors. Warthin however has recently described myocardial changes and lesions of the coronary arteries as among the most common lesions met with histologically.

Bones.—Lesions of the osseous system are among the most frequent met with in hereditary syphilis. They were recognized as early as 1747 by Rosen von Rosenstein and have been the subject of much study. In general two rather distinct types of lesions occur—the so-called osteochondritis syphilitica, to which Wegner's name is frequently attached, and an osteoperiostitis. The osteochondritis is typically found in the stillborn or very young infant and so may be said to belong to the group of fetal lesions. Periostitis may occur early, but is more frequently a "late" lesion. In addition gummata occur but much less frequently than either of the other two forms.

The osteochondritis is essentially an inflammatory process at the junction of the diaphysis and epiphysis of the long bones which leads to a failure of normal growth, and hence to a faulty calcification and ossification. Seen most frequently at the lower end of the femur and humerus and the upper end of the tibia it may involve any of the long or short hollow bones. Some authors, as Wegner, describe three types or stages of the lesion—others two and some even four. They are all part of the same general process. If the femur of a stillborn syphilitic infant be cut lengthwise it will be noted that the fine regular grayish-white epiphyseal line is broadened to a line from 2 to 6 mm. in width depending on the stage of the process. The color is yellowish and in marked cases the substance at the junction is gritty and spongy. As the line widens it becomes irregular. Histologically most complicated pictures are found depending on the age and extent of the process. Essentially there is an interference with the process of calcification, necrosis of the cartilage cells and inhibition of ossification. On the diaphyseal side of the line there is a formation of granulation tissue which may reach such a degree that the epiphysis becomes separated from the shaft and subluxated. Attempt at repair and bone formation from the periphery is usually taking place at the same time the destructive lesion is in process.

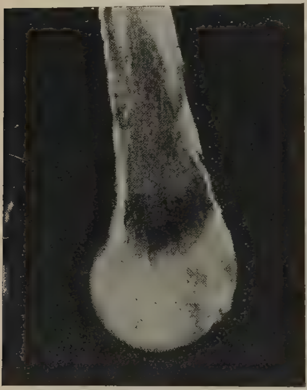

FIG. 348.—Osteochondritis showing broad zone of granulation tissue and irregular epiphyseal line.

Periostitis in infancy is more apt to be at the ends of the bones—in older children near the center of the shaft. In young children it is more apt to be quite general and very often involves the phalanges. In older children the tibia is the common seat. Periostitis of infancy rarely "breaks down" or becomes secondarily infected, but not infrequently the periostitis of the "late" type leads to a skin involvement and sinus formation. Both the periostitis of the cranium and gumma of the skull produce abnormalities in the shape of the head.

Skin, Mucous Membranes, etc.—Although lesions of the skin and mucous membranes are very common and are most important from a diagnostic standpoint, from a pathological point of view they show little characteristic and hence will not be described in detail. They are most protean in character. The catarrhal inflammation of the nasal mucous membrane, so constantly present in early infancy, may produce ulcerations with destruction of the nasal cartilages and bones and thus lead to the "saddle nose" deformity seen in later childhood. The ulcerations produced by moist lesions about the mouth and anus may end in scar formation of diagnostic importance after the active lesions have long disappeared.

Syphilitic alopecia is of little importance from the standpoint of pathology or diagnosis. Paronychia is common but probably is only a secondary result of the low resistance of the syphilitic infant. Dystrophic changes of the finger nails are not unusual.

Lymphatic Glands.—Although one of the most constant results of a syphilitic infection, the almost universal adenopathy shows nothing specific from a pathological standpoint. The hypertrophy is uniform, limited in degree, and the enlarged glands do not undergo degenerative changes or suppuration. Gummatous change may occur and we have seen extensive ulceration result. This is very uncommon compared with the general adenopathy.

Central Nervous System.—One of the most frequent and most important manifestations of hereditary syphilis is the involvement of the central nervous system. Not only are changes frequently found at autopsy, but Jeans has shown that approximately one-third of both infants and older children with lues have changes in the spinal fluid signifying an involvement of the central nervous system—a figure much the same as in adults with acquired syphilis. In infancy the so-called serous meningitis is not at all infrequent with a resulting hydrocephalus. In addition pachymeningitis hemorrhagica occurs in infancy. Various localized and diffuse forms of sclerosis and perivascular infiltration are seen in both infants and older children. Both perivascular changes and cellular degeneration in the spinal cord have been described but cord changes are much less frequent than cerebral. Lesions of the peripheral nerves have not been described. We now know that the so-called "parasyphilitic" lesions of children (juvenile paresis and tabes) are due to the direct action of the syphilitic virus and hence must be included among the lesions of the central nervous system. Basilar meningitis is very frequent with a resulting hydrocephalus.

Special Sense Organs.—With the lesions of the central nervous system may be grouped the lesions of the organs of special senses. Ocular changes are very common. In infancy iritis and chorio-retinitis are much more frequent than is generally considered. Neuritis of the optic nerve occurs rarely in both infancy and in older childhood. While chorio-retinitis may occur late, the common ocular manifestation in older children is the interstitial or parenchymatous keratitis (both terms being applied to the same condition). Green in our clinic has found abnormalities in 74 out of 100 consecutive cases. Over half showed some involvement of the eye grounds and he considers a brownish punctate pigmentation of the retina to be quite characteristic of hereditary syphilis.

Neuritis of the acoustic nerve leading to a rather rapidly developing deafness, bilateral as a rule, is seen at times in late syphilis. Although described as one of the "Hutchinson's triad" it is distinctly an uncommon lesion.

To some immeasurable degree dystrophic changes of the central nervous system may be ascribed to the syphilitic virus as the nervous system is relatively undeveloped at the time of birth compared with the other systems.

Syphilitic Dystrophies.—Fournier a number of years ago advanced the conception of "syphilitic dystrophies" which holds even today a prominent place in the discussion of hereditary syphilis by the French school. Fournier believed that without actually transmitting the infection itself from parent to offspring, a syphilitic taint in the parents exercised a deleterious influence upon the unborn child, leading to structural changes. Almost every complaint to which children are heir has been included among these syphilitic dystrophies. Many of the most striking dystrophies classified by Fournier—mental retardation, for example—we know now may be due to the actual interference of the infection. Further, there is no evidence that congenital harelip, cleft palate, etc., have any closer relationship to a syphilitic infection in the parents and grandparents than might be expected by the laws of chance and probability. Holt for example did not find a single luetic infection among 56 consecutive patients with congenital malformations and our experience coincides with these findings.

By the WR it has been shown that the apparently non-syphilitic constitutionally inferior children of syphilitic parents are actually syphilitic as a rule. We do, however, find that in the third generation dystrophic changes may occur. Thus we have seen cases of mental and physical deficiency in the non-syphilitic children of hereditary syphilitics. There are no exact studies to show the frequency of such degenerative conditions and the importance laid upon the subject by Fournier is without question very much exaggerated.

SYMPTOMATOLOGY

Infantile Syphilis.—It is exceptional to find symptons or lesions of syphilis in the newly born infant. In over 50 per cent. of our material the syphilis manifested itself in the first month of life and in another third during the second month. It is only occasionally, however, that the development of signs of the infection is delayed until after the fourth month. A tabulation of the time of development of the earliest symptoms in 95 consecutive luetic infants in our clinic was as follows:

1 week—16	1 month—51
2 weeks— 5	
3 weeks—14	
4 weeks—16	
5 weeks— 2	2 months—30
6 weeks—13	
7 weeks— 5	
8 weeks—10	
	3 months—6
	4 months—6
	6 months—1
	10 months—1
	95

Rhinitis.—One of the most common and characteristic lesions of infantile hereditary syphilis is a profuse nasal discharge. This rhinitis or "snuffles" was present in 74 per cent. of our material and was as a rule one of the earliest manifestations to appear. In the milder forms the coryza may be very slight, but not infrequently the snuffling obstructed breathing from the blocking of the nasal passages is so marked that it may be heard across the room. The discharge varies in amount but is quite constantly present, and may be very profuse and blood stained. Deep ulceration of the mucous membrane takes place at times involving the cartilage and bone. This may allow the bridge of the nose to "fall in" with the resulting "saddle nose" deformity so suggestive of a syphilitic infection. The destructive process may take place with considerable rapidity, but fortunately the deep ulcerations occur in but a small percentage of the cases. The snuffles when severe may seriously interfere with nursing as the infant cannot breathe while taking the nipple, and this may become a mechanical factor adding to the malnutrition of the syphilitic infant.

Cutaneous Eruptions.—As in the acquired type cutaneous lesions form one of the most common manifestation of an inherited syphilitic infection. In our material some form of eruption was observed in 80 per cent. of the infants. It nearly always develops during the first two months of life and is usually, although not necessarily, associated with rhinitis. Certain skin lesions are also observed later in infancy during a recrudescence of symptoms occasionally observed. Two general forms of eruptions are seen—a general and a circumscribed form.

Diffuse or General.—Not infrequently the first change to be noted is a generalized dusky-yellow color, particularly noticeable on the face, which has been likened to the color of "cafe au lait" by the French. The skin becomes glossy and the color may deepen as the lesion grows older. As the lesion progresses the eruption becomes most striking on the palms of the hands and the soles of the feet where desquamation takes place and even large layers of the epidermis become loosened and exfoliated, leaving a skin beneath which is shiny and often distinctly red or "ham" colored. In our experience this desquamation or exfoliative dermatitis involving the palms and soles is the most frequent and characteristic of the syphilides and occurred in over two-thirds of our infants with syphilis who developed an eruption. At times a group of circumscribed lesions becomes confluent and gives rise to a diffuse lesion. The infiltration into the cheeks may be the starting point of ulcerations which leave deep linear scars radiating from the mouth. These scars or rhagades seen in an older child are almost pathognomonic of an active lesion having been present in infancy.

Circumscribed Eruptions.—A number of circumscribed forms are seen in infancy which resemble very much the lesions of acquired lues. These may develop on top of a diffuse infiltration or arise independently from a normal skin. The most common of these are:

(*a*) Maculopapular syphilide. This consists of small slightly elevated spots from 0.5 to 2 cm. in diameter found most frequently

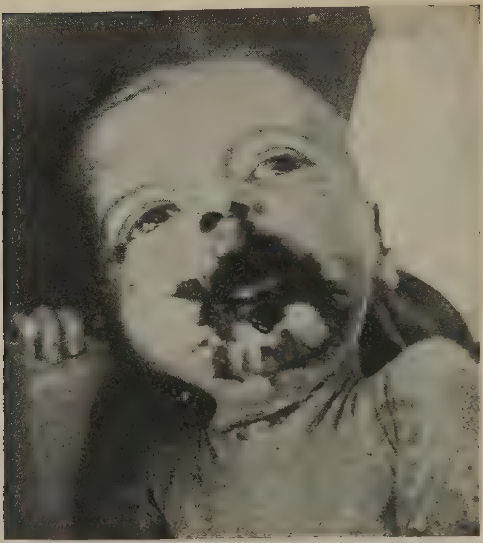

FIG. 349.—Severe infantile syphilis with snuffles and deep ulceration about mouth. (St. Louis Children's Hospital.)

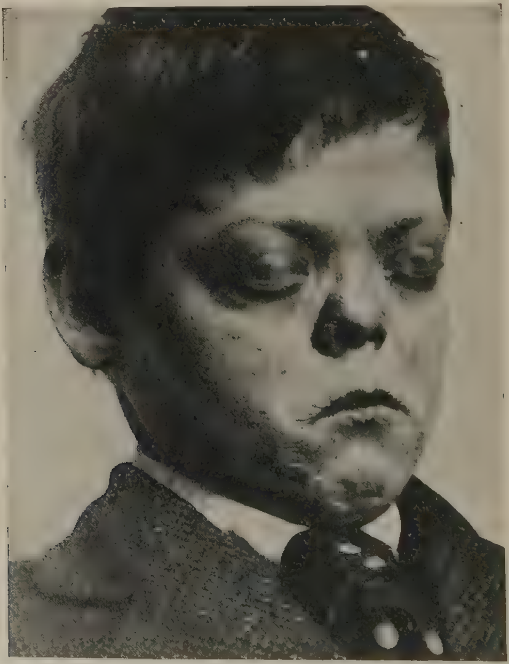

FIG. 350.—Boy of 15 years showing saddle nose deformity. Rhagades about mouth. (St. Louis Children's Hospital.)

over the extremities, face, neck, palms, and soles. The color may vary somewhat—the younger lesions being a bright coppery red color and the older more "ham" colored. These lesions may disappear without leaving a trace, or may at times desquamate or even ulcerate and crust over. At times the lesions are quite small and distinctly elevated and these at times are separately grouped and designated as a "papular syphilide." At times they are circinate (Fig. 351).

(*b*) Papulo-pustular syphilide. More rarely a papulo-pustular lesion develops in severe cases. Usually the pustules are discrete and scattered, but at times they may become confluent and in this way an extensive ulcerated crusted area may be found. The pustules become crusted and dry but at times may ulcerate. The pustular syphilide must be distinguished from the not infrequent septic or pyemic skin eruptions which are very apt to occur in hereditary syphilis as a result of the poor nutritional condition of the infant.

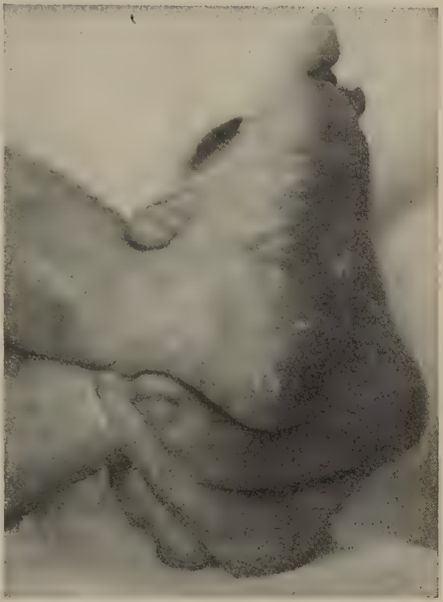

Fig. 351.—Circinate eruption in an infant. (St. Louis Children's Hospital.)

(*c*) Condylomata and Mucous Patches. In moist parts as about mouth, anus or genitals, between the thighs, etc., where excoriations of the surface are apt to take place, large plaque-like raised patches are seen with a moist surface and infiltrated walls. They are somewhat elevated but show little or no inflammatory reaction as a rule. They are in reality papular or maculo-papular lesions which have undergone ulceration following maceration of the surface. The condylomata are seen chiefly as recurrences.

(*d*) Syphilitic Pemphigus Neonatorum.—This is a form of circumscribed lesion which must be mentioned apart, as in contrast to the others it is practically present only at birth. It is a bullous eruption which may be widespread or more or less limited to the soles of the feet and palms of the hands. The vesicles are usually from 0.5 to 2 cm. in diameter, but several may become confluent forming large blebs. The vesicles are filled with a purulent or sanguino-purulent material in which the treponema can be found readily. This eruption is usually significant of a severe lethal infection.

Malnutrition.—Under-nourishment and bad nutrition is one of the most characteristic and common manifestations of the syphilitic infection in an infant. Not only is the newborn infant apt to be underweight, but the malnutrition is of a type all out of proportion to the gastro-intestinal symptoms present, and in fact these may be lacking. It is of a type that continues and progresses until specific treatment is instituted, but not infrequently babies are encountered in which the nutritional condition does not respond to treatment. In these cases, although the associated symptoms, as eruption, disappear rapidly, the wasting progresses steadily until death ensues. This malnutrition which is seemingly the result of an impairment of the functional capacity of the various organs and glands is one of the most important factors in the production of the high mortality rate of hereditary syphilis.

The extent to which malnutrition occurs in syphilis is shown by a study of the weights in a series of 77 untreated luetic infants at the time of admission to the clinic or hospital. The weight of each infant was compared with the average weight of a healthy infant of the same age and the difference noted. Of the 77 syphilitic infants seven were over weight, 10 average weight, and the remaining 60 or 77 per cent. were below the average weight. The extremes varied from 1430 gm. above to 3100 gm. below. An average of the entire series of 77 infants was 1480 gm., or a little more than 3 lb., below the average weight of a healthy infant. The ages of the infants in the series were from two weeks to five months.

Visceral Lesions.—As noted under the discussion of the pathology of hereditary syphilis, enlargement of the spleen and liver are of almost constant occurrence. Demonstration of this enlargement affords valuable clinical confirmation of a luetic infection when present in association with other signs. The exact percentage in which enlargement takes place depends entirely upon the definition of what the observer regards as enlargement. Thus, figures for splenic enlargement vary with different observers from 10 to 80 per cent. Still noted the spleen as easily palpable in 45 per cent. of his cases and enlargement of at least one inch below the costal margin in 22 per cent. We found the spleen definitely palpable in 82 of a series of 100 infants. Under six months of age there are relatively few causes for enlargement of the spleen—at least for an increase to a size which makes it easily palpable. It is in these young infants especially that splenic enlargement is of clinical significance. The rachitic spleen is later in development and other causes, such as an occasional malarial spleen or a splenic tumor, are uncommon. In our experience an enlarged spleen in an infant under four months without symptoms of an exudative diathesis usually means either syphilis or tuberculosis, and the former is of course much more common.

Hepatic enlargement as a syphilitic manifestation is always seen in conjunction with some degree of splenic enlargement. It is only of clinical significance in the young infant. As a rule the liver is not palpable below the costal margin in the infant of from one to six months of age, except in a small percentage of individuals. Hochsinger considers the liver as showing a definitely demonstrable enlargement in approximately a third of his syphilitic infants.

Enlargement of Lymph-nodes.—In our experience enlargement of the lymph-nodes in the syphilitic infant is of relatively little importance. Granting that the small shot-like polyadenitis is of syphilitic origin, it is practically impossible to differentiate it from the palpable nodes which are found in practically every undernourished infant. Hochsinger, who lays more stress on the adenitis than most observers, considers the appearance of palpable glands in places where glands are not normally palpable as of most importance, and among these the cubital and intercostal glands. In our experience the demonstration of these lymph-nodes in the syphilitic infant is rather unsatisfactory and uncertain, though gross enlargement of the epitrochlear glands is of some significance.

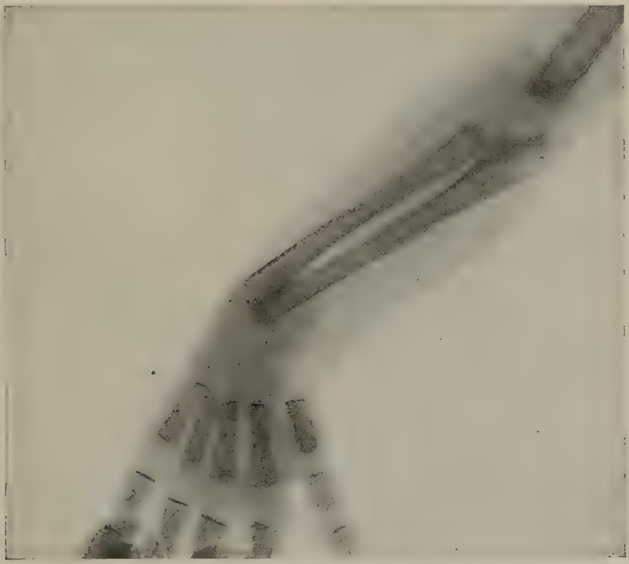

Fig. 352.—*X*-ray of osteoperiostitis and chondritis in syphilitic infant showing destruction of lower end of radius. (St. Louis Children's Hospital.)

Bones.—Lesions of the osseous system are very common. Not only do they produce definite acute clinical conditions, but certain more chronic changes in the conformation of the cranium which are quite characteristic. Our knowledge of the frequency and importance of the bony changes has been considerably increased in recent years through the development of roentgenology.

Epiphysitis (*Osteochondritis*).—The osteochondritis at the junction of the shaft and epiphysis of the long bones, described in the chapter on pathology, is one of the common hereditary syphilitic lesions. This is easily demonstrable clinically in the roentgenogram where the epiphyseal line is found irregular and widened and there may be a definite slight subluxation of the epiphysis. Some swelling of the periosteum at the epiphysis may also be noted. Although these changes are frequently present in the *x*-ray, it is only in a much smaller degree that definite clinical signs of the bone changes become apparent. Thus epiphysitis as a clinical entity—swelling, with pain on motion—developed in some 11 per cent. of Still's cases and was noted in 14 per cent. of our own. The earliest the lesion was noted in our series was in the second week and the latest during the sixth month. Not infrequently the first clinical evidence noted is the sudden loss of the use of a leg or arm, usually the arm as it is the lower end of the humerus which is most frequently involved. At times the lesion is bilateral.

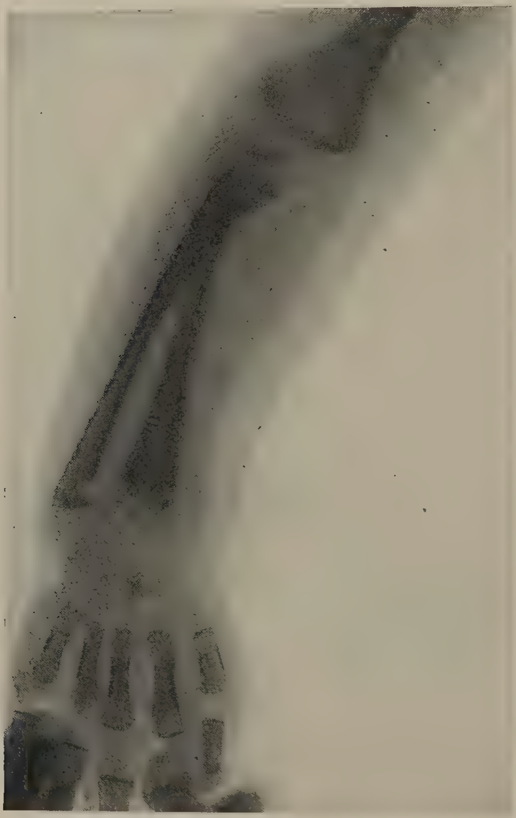

Fig. 353.—*X*-ray of osteochondritis lower end of ulna and radius. (St. Louis Children's Hospital.)

The name "Parrots pseudo-paralysis" or "syphilitic pseudo-paralysis" is often applied to the condition. The "paralysis" is flaccid in type and there is usually moderate tender swelling about the joint and acute pain on motion. The x-ray may show considerable subluxation of the epiphysis while the end of the diaphysis is flaring from a periosteal callus formation. The soft parts about the joints frequently show swelling and distention. The condition is easily distinguishable from the spasticity of a cerebral palsy by the pain on motion and accompanying swelling, and from scurvy by the early age at which it develops. The "paralysis" is protective in nature and disappears rapidly under specific treatment.

Cranial Changes.—Changes in the conformation of the skull are so frequent that they require special mention. It must never be forgotten that syphilitic infants are prone to develop rachitis, and the cranial changes with the exception of early hydrocephalus in our experience are usually the result of a combination of the two diseases. In an individual case it is very often extremely difficult to say just how much of the change is due to the rachitis and how much to the syphilis. Enlargement of the head and hydrocephalus is frequently met with. The true syphilitic hydrocephalus is the result of a meningitis and as a rule definite changes in the spinal fluid are present. It is usually limited in extent and hence is easily confused with a simple rachitic enlargement of the skull. The older view, that hydrocephalus is usually syphilitic in origin, has little support, however, as the result of recent investigation. Syphilitic bossing, as a result of periostitis, is sometimes seen in early syphilis. These are apt to be situated upon the frontal and parietal regions and are sometimes called "Parrot's nodes." As the ordinary rachitic bossing is in the same regions, here again in an individual case confusion may exist. The subject of syphilitic bossing requires further study. *Craniotabes* is often seen in the skulls of syphilitic infants. There is nothing, however, to link up these soft areas with a specific syphilitic process, and we know that craniotabes is found in rachitis uncomplicated with syphilis. At times the late union of the sutures, together with the frontal and parietal overgrowth, produce the boat-shaped or "natiform" skull. This again is seen in uncomplicated rachitis. When, however, localized irregular protuberances or depressions are seen in the skull as the result of a localized osteitis, these changes are almost pathognomonic of syphilis. We have mentioned the saddle nose deformity of the face which in the syphilitic infant tends to throw even slight bossing of the frontal eminence into marked relief and accentuates even a moderate syphilitic hydrocephalus.

Dactylitis.—Although the roentgenogram has shown that osseous changes may be very widespread without producing definite clinical lesions apparent to the eye, and that involvement of the phalanges is very common (a proliferating osteitis), dactylitis as a clinical entity is less frequently met with than epiphysitis. When it develops it appears as a fusiform swelling of one or more of the proximal phalanges of the fingers as a rule. It may involve a distal phalanx or the toes, and not infrequently it is bilateral but not necessarily symmetrical. The lesion is painless and only rarely does the skin become involved and destruction of tissue take place. The metacarpal bones are rarely involved clinically, although it is not unusual to find a change in one or more of the metacarpal bones when a roentgenogram is taken of the hand when dactylitis is present. The x-ray shows a broader denser shadow of the afflicted bone as a result of the osteoperiosteitis. Dactylitis may begin very early and in at least half of the cases is present by the third month. Tuberculous dactylitis is rarely present before the sixth month. In our experience dactylitis in children over six months is much more apt to be tuberculous than syphilitic in origin.

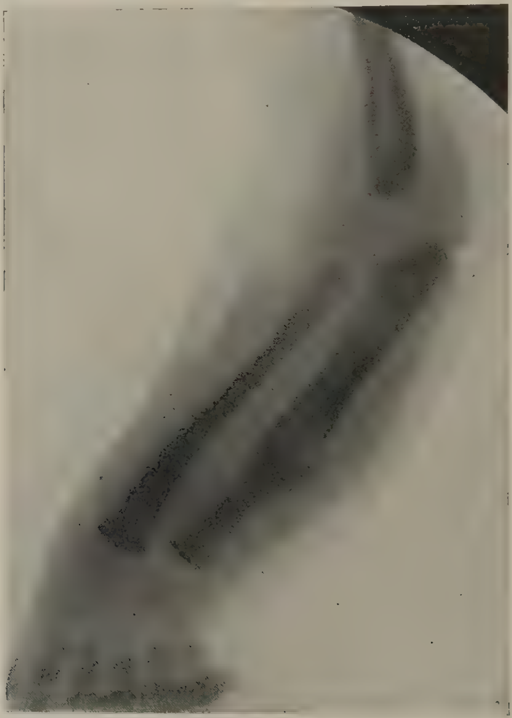

Fig. 354.—Syphilitic osteoperiostitis of ulna in an infant. (St. Louis Children's Hospital.)

Osteoperiosteitis.—We have noted the involvement of the periosteum at the epiphysis in a previous paragraph. Rarely osteoperiosteitis of the shaft of the long bones, which is the typical bone lesion of late hereditary syphilis, is seen in early infancy (Fig. 354).

Anemia.—The blood of the syphilitic infant always shows some degree of anemia. There is nothing in the type of anemia which is at all specific or characteristic or which differentiates it from the anemia found in infants with other infections or in infants with impoverished nutrition. At times nucleated reds and abnormal cells are found. It may be simple anemia or a chlor-anemia. Older syphilitic infants occasionally show the rachitic pseudo-leukemia infantum.

Less Frequent Lesions.—*Laryngitis.*—The hoarse, harsh, strident cry of the syphilitic infant has been noted by many syphilographers and is considered by some to be a characteristic symptom. It is only of diagnostic value when present in association with other more definite lesions.

Orchitis.—An involvement of the testicles in a young infant is quite specific for syphilis. Although we have rarely seen orchitis, Still found it in from 2 to 3 per cent. of his material. The testicle becomes from two to three times its normal size and often the enlargement is bilateral. It occurs in infants of a younger age than are found developing tuberculous orchitis.

Nephritis.—We have noted in the section on pathology the frequency with which pathological lesions are found in the kidney. Much less frequently clinical signs of a nephritis are noted. When present the findings are those of an acute interstitial type of nephritis with early generalized edema.

Hemorrhage.—Occasionally, widespread diffuse hemorrhages are observed in the newborn syphilitic infant, but melena neonatorum is by no means necessarily luetic in origin. At times the hemorrhages are seen associated with jaundice. Bleeding from the umbilicus sometimes occurs and may be looked upon as the result of changes in the structure of the blood-vessels.

Onychia and Paronychia.—Involvement of the nails with a dry atrophy—onychia sica—is sometimes seen. More common, however, is an infection about the nails, which has the appearance of a pyogenic infection.

Alopecia.—Just as in acquired syphilis, alopecia is sometimes seen. This is of questionable diagnostic value as the character of the hair in the infant varies so much normally. A loss of hair over the anterior part of the skull is suggestive. Some consider a loss of the eyebrows as significant of a luetic infection. Still, on the other hand, calls attention to an abundant crop of hair in the newborn as a condition not infrequently seen in lues.

Ascites.—Ascites, secondary to a cirrhotic process in the liver, has occasionally been observed in the very young infant.

Jaundice.—Jaundice in the newborn in the vast majority of instances is the simple jaundice neonatorum. Rarely it persists in the syphilitic infant and has been found in these instances to be the result of interstitial hepatitis.

Ulcer of the Umbilicus.—A slow indolent ulcer of the umbilicus in an infant under one month has been described several times. The ulcer may be accompanied by other symptoms, but it has been noted at times before the development of other lesions.

Recurrences.—After the early lesions have disappeared, either spontaneously or as a result of treatment, a recurrence is at times noted. The reaction is usually seen in the sudden development of a rather definitely localized skin eruption, which characteristically takes on the form of condylomata about the anus or genital regions. Plaques on the buccal mucous membranes and tonsils are seen at times. Considerable periods of time may elapse in some cases between the primary eruption and the recurrence. The recurrent lesions are especially amenable to treatment.

The Latent Period.—The latent period or stage of hereditary syphilis forms one of the most interesting and most obscure chapters in the story of the disease. While it has been known for years that apparently healthy children of four or five years of age or more would suddenly and for some unknown reason develop typical lesions of late hereditary syphilis, the deduction that these children were entirely free from evidences of the disease before they were brought to the physician was largely a matter of inference. With the discovery of the WR a means was first afforded to carefully study syphilitic families, and by carefully examining all the members of a family when one case was discovered, we soon began to find children of normal development and apparently free from any clinical evidence of syphilis who nevertheless gave a positive WR. Subsequently some of these children developed typical manifestations such as keratitis. Moreover, it is possible in some of these children to provoke the development of clinical lesions by the installation of vigorous treatment. It is only in a small percentage of the apparently normal children, however, that the infection is absolutely latent. If the children are carefully studied such lesions as unequal pupils, etc., can frequently be demonstrated, and in these the infection is obviously present and is quiescent rather than latent. We do, however, encounter the development of some such lesions as syphilitic arthritis in early childhood in individuals who have been carefully watched through infancy and in whom there have never been any infantile lesions, nor does the most minute physical examination disclose any other evidence of syphilis. These children give strongly positive WR reactions and the lesions respond to specific treatment. What brings about this sudden outbreak of clinical manifestations, or where the focus of infection has rested all these years are questions that at present are unanswerable.

Late Syphilis.—There is considerable analogy between the lesions of tertiary or quarternary acquired syphilis and the so-called "late" or "tardy" lesions of hereditary syphilis. Practically all of the lesions belonging to late acquired syphilis can and do occur in "late" hereditary syphilis, although some, as the cardio-vascular changes, are exceedingly uncommon in childhood. The division into early and late is not absolute so far as age is concerned, as we have noted before.

We frequently observe the development of late lesions in infants under one year of age, and in roughly 25 per cent. of our material the lesions of late syphilis have developed before the fifth year. Practically any organ or structure of the body may be involved in late syphilis. In our material in the order of frequency the structures commonly involved clinically are the nervous system, eyes, bones, joints, and the mucous membrane of the nose and throat. The majority of our patients on admission have been between 5 and 10 years of age and it is uncommon to find the development of active lesions delayed until the twelfth year or after.

The following table of 100 consecutive admissions with late syphilis gives a fairly comprehensive view of the frequency with which the different lesions are encountered and at the same time some idea of the protean character of the disease. Thirty-two of the 100 children presented lesions of the central nervous system.

Table 2

Bones:	
Periostitis tibia	4
Periostitis skull	1
Osteomyelitis	1
Joints:	
Acute arthritis knee	8
Acute arthritis ankle	1
Acute arthritis elbow	1
Chronic synovitis, knee	1
Skin:	
Gummata	3
Alopecia	3
Eye:	
Interstitial keratitis	25
Choroiditis	1
Ear:	
Deafness	1
Ulcerations:	
Nasal	2
Laryngeal	1
Pharyngeal	1
Central Nervous System:	
Cerebrospinal syphilis	14
Hemiplegia	6
Epilepsy	5
Spastic paraplegia	4
Old hydrocephalus	2
Optic atrophy	1
Mental deficiency	16
Miscellaneous Conditions:	
Ozena	1
Enlarged spleen (only symptom)	1
Enlarged epitrochlear glands (only sign)	1
Torticollis	1
Aortitis	1
Obscure abdominal pain	1
Obscure pain in legs	2
Endarteritis obliterans	1
Paroxysmal hemoglobinuria	1
Raynauds disease	1
Hutchinson teeth	4
Enamel defects of teeth	2
Mental deficiency (neg. spinal fluid)	7

Eye.—Some type of involvement of the eye is by far the most common manifestation of late hereditary syphilis. Two great groups of lesions exist—one a group related to and significant of a lesion of the central nervous system and including such conditions as strabismus, nystagmus, optic atrophy, choked disc, ocular palsy, and pupillary changes; the second, a group of inflammatory reactions situated in the eye itself as keratitis, or chorioretinitis.

The most frequent specific lesion is an interstitial keratitis or parenchymatous keratitis. It occurs in approximately a fourth of the late cases. While not absolutely pathogonomonic, it is exceedingly significant of a syphilitic infection. As a rule it develops between the sixth and tenth year, but we have seen it in a syphilitic infant of three months. It is exceedingly persistent and shows a marked tendency to recurrence. Not infrequently both eyes are involved, either simultaneously or at different times. In the acute stage the red injected eye is usually accompanied by considerable photophobia. Later considerable cloudiness and haziness of the cornea may persist. The lesion is exceedingly intractable and resistant to treatment and

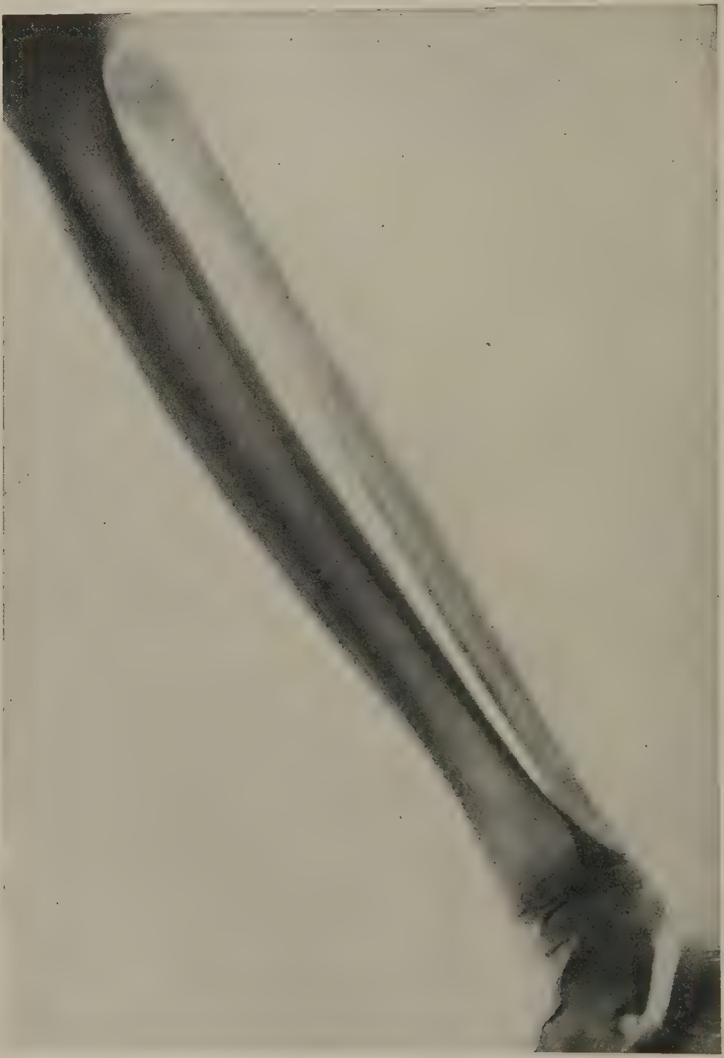

Fig. 355.—Osteoperiostitis of tibia. (St. Louis Children's Hospital.)

not infrequently we have noted sudden intensive treatment apparently bring out a keratitis or seemingly aggravate an already existing one. In a way interstitial keratitis may be considered as the most characteristic lesion of late syphilis and Hutchinson noted a frequent association between keratitis, notched teeth and deafness, which has become known in literature as "Hutchinson's triad." Choreoretinitis is not infrequently found and very rarely a gummatous iritis.

Bones.—The bones are frequently involved in late syphilis. The typical lesion is a chronic bilateral hyperplastic periosteitis of the tibia. It may develop early but it usually does not appear until the fifth or sixth year, and while generally seen in association with other lesions, it may be the only evidence of the syphilitic infection. As ordinarily seen the onset is insidious and is accompanied by little pain. On examination the bones are found thickened but have a fairly smooth surface. The *x*-ray shows strikingly the almost regular symmetrical fusiform enlargement throughout the entire length of the tibia. It is this type of lesion which leads to the anterior bowing or the so-called saber shin. In a second type the hyperplastic process is not so uniform, but is irregular in distribution and the shin bone becomes rough and the crest of the tibia becomes nodular. Necrosis may take place but usually the disease becomes arrested before this occurs, and only a tibia with a roughened nodular edge persists.

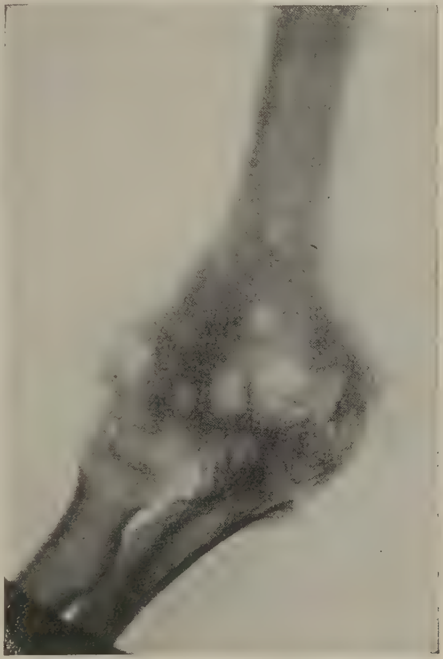

FIG. 356.—Late syphilis. Ankylosis as result of an old syphilitic arthritis. (St. Louis Children's Hospital.)

Not infrequently gummatous changes are seen and these frequently degenerate and produce sinus formations. While the tibia is the common site of gummata, the small bones of the hands and feet and not infrequently the bones of the skull are involved.

Joints.—About 10 per cent. of the children with late syphilis show some involvement of the joints. It is usually associated with other lesions as keratitis, but we have seen it develop as the first clinical symptom of lues in a child of five in whom the most careful examination failed to reveal any other lesion, excepting a strongly positive WR.

Although several types have been described, the classification into types is very uncertain and unsatisfactory. There may be a simple hydrops, involving chiefly the knees. There is little pain with the effusion and the chief complaint of the child is usually difficulty in walking. It is only very rarely that the acute tender, swollen joints as in acute rheumatic fever are found. In another type there is some erosion of the cartilage and a thickening of the synovial membrane and villæ accompanying the effusion. Impairment of function is more marked with this type. A third variety resembles more an arthritis deformans with involvement of the small joints and a chronic effusion together with checking of the capsule. There is relatively little pain and limitation is chiefly mechanical. The most characteristic thing about the syphilitic joint involvement is the tendency for a symmetrical synovitis to develop in an insidious way with a freedom from acute pain and loss of function, not seen in a joint involvement due to any other acute or chronic condition. The knees are involved in at least three-quarters of the joint cases.

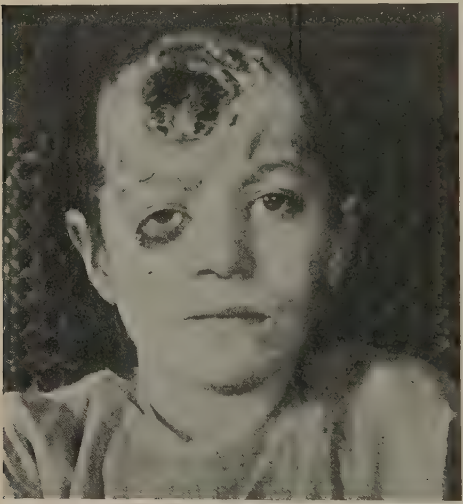

Fig. 357.—Late syphilis. Gumma of skull and face. (St. Louis Children's Hospital.)

Skin and Mucous Membranes.—Lesions of the skin and mucous membranes are largely gummatous in nature. The most common skin lesion is a fairly large tumor starting usually in the subcutaneous tissue of the thighs, which shows a distinct tendency to break down and leave a large indolent ulcer. Other regions as the face are occasionally involved. The tumor is indurated as is the ulcer if it subsequently develops. Small nodular syphilides about the size of a pea are occasionally encountered which have a marked tendency to undergo necrosis and ulceration. As a rule the skin manifestations respond readily to treatment.

Lesions of the mucous membrane are usually gummata, which undergo rapid degeneration and ulceration. They occur usually in the soft palate and pharynx and at times are seen in the larynx and trachea or on the epiglottis. The ulcers are usually painless and have sharply defined indurated borders and leave a firm white scar with considerable contraction on healing. Perforations of the soft palate are practically always the results of gummatous ulcerations.

Alopecia is not infrequently encountered and alopecia in children is always suspicious of syphilis.

Deafness.—While deafness as the result of an inherited infection has long been known it is by no means a frequent lesion. It is usually due to involvement of the VIII nerve and as a rule is bilateral. It usually begins after infancy and is rather sudden in its onset. Deafness may also be the result of a chronic osteitis leading to the occlusion of the cavities of the internal auditory meatus and the bony labyrinth by proliferating bone. Depending upon the degree of exudation vertigo may be almost absent, or at times may be very marked and accompanied by tinnitis. Central deafness may also occur.

Lymph-nodes.—While the lymph-nodes are usually enlarged, it is rare to find this of clinical or diagnostic importance. Rarely degeneration and ulceration of the lymph glands is noted and it is then to be considered as a gummatous change rather than the usual adenitis of a chronic ordinary infection.

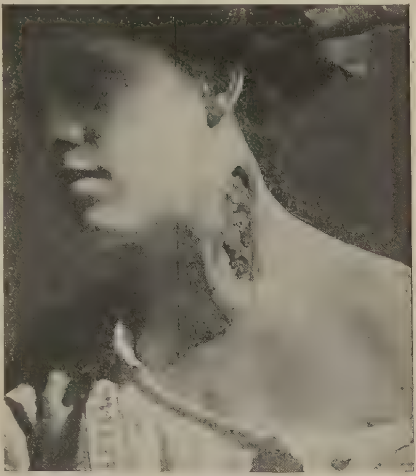

Fig. 358.—Late syphilis. Gumma of cervical lymph nodes. (St. Louis Children's Hospital.)

Teeth.—One of the best known and most striking lesions of "late" syphilis is the so-called "Hutchinson's teeth," or a deep grooving or notching of the upper central incisors of the second dentition. It is not a valuable symptom from the standpoint of frequency as we

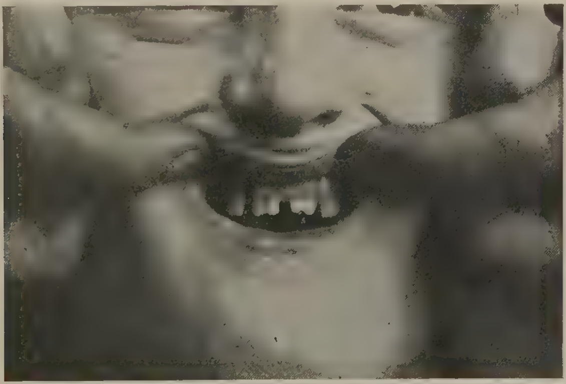

Fig. 359.—Hutchinson teeth. The separation and irregularity are as characteristic as the notching of the upper central incisors. (St. Louis Children's Hospital.)

find it in but 6 per cent. of our late cases. Furthermore, Hutchinson's teeth may rarely occur in other conditions when there has been an early interference with the formation of the teeth from some other cause, and hence is not absolutely pathogonomonic of syphilis as so frequently considered. This peculiar notching, which is in the permanent teeth and hence not seen until after the fifth or sixth year, is the result of changes in the development of the tooth bud early in infancy. Thus the so-called late lesion is actually an infantile change. Much more commonly we find badly formed, irregular, pegged shape teeth, irregularly spaced and uneven as the result of interference with dentition. These changes are found not only in the permanent teeth but also in the teeth of the first dentition.

Cardio-vascular.—The cardio-vascular system is only rarely involved in hereditary syphilis which is one of the most striking differences between the hereditary and acquired infections. Aortitis rarely occurs and syphilis is the cause of the few cases of non traumatic aneurysm which have been described in childhood. Arteriosclerosis is very infrequently met with. Endarteritis is occasionally encountered. Most frequently it is a part of a cerebro-spinal syphilis, but we have seen a case of symmetrical gangrene of the extremities as a result of an endarteritis obliterans.

Raynaud's Disease is not especially rare in connection with hereditary syphilis. The exact etiology and pathology are unknown, but it is the opinion of many that the condition may be due to syphilis.

Paroxysmal hemoglobinuria has been repeatedly described in relation to hereditary syphilis. It is quite rare but seemingly pathogonomonic of lues. The exact pathology is not yet clear. An autohemolysis occurs following exposure to cold which is accompanied usually by a chill and fever. The amount of blood destroyed is small and the free hemoglobin is rapidly excreted in the urine. One of our cases had series of repeated attacks followed by long intervals of freedom.

Visceral Lesions.—Visceral lesions in late syphilis are usually gummatous in character. The liver is the most frequent seat and next the spleen. Hypoplastic changes may also occur. Chronic interstitial nephritis due to syphilis is occasionally encountered. Gumma of the adrenals, testes, and other glands have been described. Diabetes insipidus has been noted in children with a syphilitic infection, but the pathogenesis is very obscure.

Muscular Pain.—Although no typical lesion or involvement of the muscles has been described, we have encountered a number of cases of obscure muscular pain, torticollis, and the like, which have failed to respond to the usual treatment. In a number of these cases the WR has been found positive and immediate relief and disappearance of the symptoms followed specific treatment.

Central Nervous System.—The rather prevalent opinion that hereditary neurosyphilis is relatively uncommon seems to us entirely unfounded. Involvement of the central nervous system occurs in hereditary syphilis with the same frequency as in acquired syphilis, though with a difference in the frequency of certain clinical manifestations. One reason for the current opinion may be that casual observation is not as effective as observation with a purpose. Another difficulty is that the newer laboratory methods of diagnosis have not been sufficiently applied to this group.

Though no proof has been brought forward, it seems reasonable to presume that, during the general invasion of the body by the treponema, every organ and tissue, including the nervous system, is invaded. Treponemata are known to exist in the blood stream in the early stages of the infection and it is difficult to see how the nervous system can escape. It is equally logical to assume that in many instances the infection undergoes spontaneous resolution just as it does in the skin and elsewhere, perhaps never to recur. In some instances it may manifest itself clinically and in others assume a subclinical activity only to be openly manifest at a later period. The earliest activity is meningeal. Clinically this is manifest by the usual meningeal signs. The cerebrospinal fluid exhibits increased cells, albumen, and globulin and gives a positive Wassermann reaction and colloidal gold reaction. The cerebrospinal fluid in cases with subclinical activity shows the same pathology though often in less degree. In our clinic slightly more than one-third of the syphilitic infants have shown sufficient pathology in the cerebrospinal fluid to warrant the conclusion that an infection was present in the meninges at the time of examination. But one-third of those with pathological cerebrospinal fluids had clinical evidence of neurosyphilis. Similar examination of a group of older syphilitic children revealed the fact that about one-third had cerebrospinal fluids sufficiently pathological to warrant a diagnosis of neurosyphilis and that two-thirds of the neurosyphilitic cases had clinical evidence of their nervous system infection. It is probable that the late group with pathological cerebrospinal fluid represents a continuation of a group entirely similar to the early group; i.e., all who develop neurosyphilis at any time have had continuously from birth an infection of the nervous system with a pathological cerebrospinal fluid. The older such children become the greater the proportion who show clinical evidence of their neurosyphilis.

Meningo-vascular Lesions.—The early lesions of neurosyphilis are meningeal or meningo-vascular. As a result convulsions or hydrocephalus or both may occur. The convulsions of syphilitic meningitis may be repeated numerous times daily or may be separated by intervals of weeks. They are usually afebrile. Sometimes a bulging fontanelle and general neck rigidity without convulsions may be found. Syphilitic meningitis has been much more common in our material than the literature on the subject would indicate in regard to the general incidence. The cerebrospinal fluid pathology resembles that of tuberculous meningitis except for the Wassermann reaction and the colloidal gold curve. Albumen and globulin are increased and the cells may be from 100 to 300 or more per cubic millimeter. Increase in the amount of fluid is the rule and not infrequently the increase is great enough to cause hydrocephalus. The hydrocephalus is not necessarily of the internal variety but may be a generalized increase with little or no change in the size of the ventricles. Syphilitic meningitis clinically manifest in a child old enough to make the determination of mental impairment possible usually causes a marked degree of deficiency. Under specific therapy the most surprising recovery of mental capacity may occur. On the other hand the results may be most disappointing in this respect, there having occurred apparently an arrest of cortical development. As the inflammatory process extends more deeply into the cortex or into the cranial nerves various paralyses or sensory nerve involvements are seen. Our experience has been that *hemiplegia* is the most frequent variety of acquired paralysis due to syphilis. Also that 50 per cent. of our hemiplegias were due to this cause. Pathologically the lesion is an encephalitis associated with an arteritis and differs anatomically and histologically little or none from other forms of encephalitis. Acquired paraplegia may also occur as a result of syphilis. Paraplegia occurring as a manifestation of late syphilis is sometimes of the spinal or flaccid type though cerebral symptoms are usually associated. The pathology is similar to that of cortical involvement. The myelitis may be circumscribed or diffuse and is associated with an endarteritis. The various paralyses occur most frequently late in the secondary period or early in the tertiary period of the disease. Our youngest hemiplegic child was observed at 18 months. Optic nerve involvement occurs at about the same period though we have observed several infants with optic neuritis under the age of three months. Affection of any of the cranial nerves is possible and has been reported as occurring, but in our experience with the exception of the optic nerve this event has been uncommon. Hutchinson recorded 41 cases of deafness from syphilis in his 60 years of observation. It has fallen to our lot to observe but one recognized case in an eight year period of fairly intensive observation. Hutchinson noted the preponderance of females among the syphilitic deaf and also the fact that deafness occured after the period of infancy.

Epileptiform seizures differing in no way from "idiopathic" epilepsy are common as a result of hereditary syphilis. Treatment early in the disease usually results in a clinical cure, but treatment at a later period often gives little or no relief even though there occurs a serologic cure. In this connection it is necessary to point out that a Wassermann survey among adult epileptics cannot show the frequency of hereditary syphilis as a cause.

Parenchymatous involvement of the nervous system is rather frequent in children but it does not commonly take on the definite clinical picture of tabes and paresis. We have observed but one case of definite tabes, and that in a girl of 18 years. Patients showing diminished reflexes and pupillary changes have not been unusual. Ataxia, disturbances of gait, and Romberg's sign are uncommon. Absence of patellar and Achilles reflexes is said to be not so constant in juvenile as in adult tabes. The Argyll Robertson pupil is considered a cardinal symptom. Optic atrophy is present in a large percentage of cases and is usually the earliest symptom. The first signs usually appear late in childhood though cases have been reported as early as five or six years. The occurrence of tabetic symptoms as a result of hereditary syphilis has been recorded as being first observed as late as the fortieth year.

Paresis.—Paresis in children is observed somewhat less infrequently than tabes. The onset is usually at 12 to 16 years or later. Cases have been reported at eight years and under but it is doubtful if it can be diagnosed so early. The characteristic symptoms are both psychic and somatic. In a young child the intellect is not sufficiently developed to show the psychic changes. A positive diagnosis could scarcely be made on the somatic changes which are merely those of syphilis. What is occasionally observed in the child is a syphilitic dementing process which sets in after three or four years and is rather rapidly progressive. A child previously of average intellect up to the age of onset may become a helpless idiot in the course of two or three years. Such cases cannot be classified as general paresis though they may be closely allied. The main symptoms of juvenile paresis, especially as differing from adult paresis may be summarized as follows: The face becomes expressionless. There are automatic sucking, smacking and chewing movements and humming which rarely appear in the adult. Pupil changes are present early, but they may be merely sluggish instead of fixed. A special peculiarity is the early and frequent occurrence of epileptiform seizures, which have been spoken of as paralytic attacks. The psychic symptoms frequently have a sudden onset. There is a progressive defect in intelligence and judgment, without fixed ideas or hallucinations. Ideas of grandeur, when present assume an infantile character. The child becomes apathetic, undemonstrative and gradually forgets all that has been learned. Occasionally there are outbreaks of passion but mania never occurs. There are increasing disturbances of speech, the vocabulary diminishes and speech finally ceases, except a few unintelligible words. Gradually the somatic symptoms become more pronounced, the child becomes bedridden and death results from pneumonia, a paralytic attack or sepsis originating in decubitus. Remissions, though common in adults are rare in children. The duration of the disease is usually from three to five years or even longer. The pathology of juvenile paresis differs but little from that of the adult type except possibly the greater frequency of cord lesions in the former.

Circumscribed **gummatous formations** in the brain are rare and none has been diagnosed in our material. They are said to result in intense headache, occurring chiefly at night and to lead to epileptic attacks and paralyses. Brain tumor symptoms may also be caused by encephalomalacia or a circumscribed meningo-vascular process.

Just as somatic syphilis may remain in such a state of relative inactivity that it may be diagnosed only by a Wassermann reaction, so may a neurosyphilis be diagnosed only by a pathological cerebrospinal fluid. It is by the recognition and treatment of such cases that much hardship may be saved the patient and his social usefulness retained. It has been our observation that 20 per cent. of all syphilitic infants and 10 per cent. of older syphilitic children have neurosyphilis in a latent form. This point is of sufficient importance that it well may be illustrated by a case. D. W. a boy of 10 years. The father is said to have had syphilis. The boy had snuffles and an eruption in early infancy. There was no present complaint, the boy being brought for a health examination. The examination showed him to be well developed and nourished. The head was large (circumference 22½ in.), the forehead high and protruding. The upper central incisors were widely spaced, pegged and centrally notched. There was a disseminated choroido retinitis on both sides—a circular area of light colored material surrounded by heaped up pigment. There were no abnormal neurological findings. The speech was rather rapid and a bit slurring at times. The mental age was in excess of the chronological age by 1½ years. Psychopathic examination revealed no abnormalities and included inquiry into memory, judgment and sociological reactions. The Wassermann on the blood serum and on the cerebrospinal fluid gave complete fixation with non-cholesterolized antigen, using 0.4 c.c. in the case of the cerebrospinal fluid. The cell count in the fluid was 187. In this case there can be but little doubt of active infection of the nervous system and yet the consulting neurologists could find no clinical evidence. Except for the syphilitic teeth this boy might easily have passed as a normal boy both physically and mentally.

Mental Deficiency.—The rôle of syphilis in the causation of mental deficiency has been under discussion for many years and a satisfactory statistical statement is still impossible. Certain it is that syphilis may be the sole cause of mental defect and it is our observation that fully one-half of the older children with clinical neurosyphilis have gross and unmistakable deficiency. On the other hand a few neurosyphilitics are intelligent beyond their years. Taking all cases of mental deficiency in childhood we find the number due to syphilis is relatively small but among the late syphilitics our records show that more than one-third have at least mild degrees of this disorder. When occurring as a manifestation of late syphilis it is more often found associated with other symptoms, such as fixed pupils, paralysis or a complicated brain syphilis. Numerous studies of the WR reaction in children in institutions for defective children have been made with most astonishing differences. What is important is not the number of mentally deficient children with syphilis, but the number of syphilitic children with mental deficiency.

DIAGNOSIS

The diagnosis of hereditary syphilis is based upon the clinical manifestations and laboratory findings. As a rule the diagnosis is not difficult if the protean character of the disease is kept in mind. In fetal syphilis there is frequently the history of repeated abortions. In a suspected case an incision through the lower end of the femur of the fetus will disclose the presence of the broad yellowish line of an osteochondritis if the infant is syphilitic. In the young infant the picture of acute infantile syphilis is quite characteristic. The combination of snuffles, eruption, and enlarged spleen is quite the constant picture. The eruption is not difficult to distinguish from intertrigo with which we have occasionally seen it confused. Most suggestive of syphilis is the exfoliation of the skin on the palms of the hands and soles of the feet. An acute epiphysitis must be distinguished from a scorbutic joint at times. One of the most important things to be kept in mind is the early development of the lesions in infancy. Lesions similar or suggestive of syphilis but due to other causes usually occur in infants over three months of age rather than in the young infant. As an absolute rule any suspicion of syphilis should be confirmed with a WR. Should the reaction be positive the diagnosis may be considered as established. Should the WR be negative and the clinical condition still be suspicious of syphilis a second and even a third test should be made after an interval of time, as in very young infants with syphilis a negative WR is occasionally found. In our own experience the WR later becomes positive in these circumstances unless treatment is established in the stage of a negative WR.

A further and absolute means of diagnosis is the finding of the treponemata by dark field. Serum expressed from a denuded secondary lesion will show treponemata in an untreated case at practically every trial.

The latent period is most difficult of diagnosis. In many cases there is no early history of symptoms or lesions suggestive of syphilis. The majority of cases of latent syphilis we have seen have been found by examining the apparently healthy brothers and sisters of known cases of lues. Careful clinical examination of these children will sometimes show stigmata of infantile lues as Hutchinson or peg teeth, rhagades, or saddle nose. Or signs of an inactive syphilis of the central nervous system, as fixed pupils, mental retardation, etc. will be found. Not only does the blood give a + WR but pathological changes in the spinal fluid will be found at times. We have seen many of these so-called latent cases subsequently develop the lesions of late syphilis.

Late syphilis may be either easy or difficult to diagnose from the standpoint of clinical findings alone. Keratitis, certain types of arthritis, tibial periostitis, fixed and unequal pupils, etc. suggest syphilis immediately. On the other hand the origin of certain symptoms, as obscure muscular pain in a child without history of infantile syphilis or syphilitic stigmata, will not be discovered unless a WR is made. In untreated late syphilis a + WR occurs in practically 100 per cent. of the cases.

Enough has been said regarding the value of the WR. The luetic reaction of Noguchi has been found to be without value and to have given considerable misleading information. In our opinion it should never be used.

Whenever syphilis in a child has been diagnosed a complete neurological examination of the child should be made for it is the lesions of the central nervous system, as blindness, mental deficiency, and hemiplegia, which make hereditary syphilis of such importance and cost to the community. The examination should include an ophthalmoscopic examination as well as serological examination of the spinal fluid. The most important change found is a + WR. Less important are increased cells, globulin and albumen. The colloidal gold reaction is important and confirmatory but not of the same significance as a + WR.

PROGNOSIS

The prognosis in infantile syphilis depends upon several factors. The more severe the infection and the earlier its manifestations the higher the mortality. Also the longer the disease exists before treatment is begun the more likely is the result serious or fatal. Fortunately, the severe and very early manifestations are in the minority. The average syphilitic infant when properly treated has not only a good chance for life but also for health and freedom from further development of syphilitic manifestations. Most of the deaths are associated with nutritional disturbances and the most important controllable nutritional factors are human milk and care not to start the treatment too intensively. The next most serious event to death is involvement of the central nervous system. Though many have laboratory evidence of neurosyphilis, but few have serious irreparable damage early in infancy. The remainder respond surprisingly well to prompt energetic treatment. Any other events are of relatively minor importance.

Unfortunately, it has been and is the habit of many to treat syphilitic infants only until the apparent signs and symptoms have disappeared. This attitude is due in some quarters to indifference. Hutchinson defended such treatment by the statement that eruptions which disappear under treatment seldom recur and that the administration of mercury to infants seriously affected the soundness of the permanent teeth. If it is granted that the teeth are made inferior by continued treatment it must be admitted at the same time that such a condition is preferable to some of the more serious consequences that may be observed in the tertiary stage. It is well established that treatment only to the relief of symptoms leaves the patient susceptible to the tertiary manifestations. An apparently complete cure is much more readily accomplished in the case of an infant than in the case of an older child. As well as can be judged in the period since the use of the newer remedies and the more recent method of application infants who are well treated do not later have any manifestations whatever of syphilis and enjoy as good health as though they never had had the disease.

As a direct result of late syphilis death but rarely occurs. The most serious event is irreparable damage to the central nervous system to which very often death is preferable. Of our own material more than 10 per cent. of the late syphilitics were in this manner rendered practically useless or worse than useless members of society. Some neurosyphilis at this stage is amenable to treatment but not optic atrophy, paralysis or dementia. The management of such conditions is best carried out prophylactically by adequate treatment in infancy. Compared to neurosyphilis all other common lesions are of minor importance, since they all respond well enough to treatment and in general do not permanently impair the usefulness of the individual.

TREATMENT

The easiest, surest and best manner of managing hereditary syphilis is, when possible, by the prophylactic treatment of the parents. It has already been pointed out that every mother of a hereditary syphilitic child is also infected and that the infection of the fetus takes place by way of the placenta. It has been established that anti-syphilitic treatment of such a mother during the time of her pregnancy will result in non-syphilitic offspring. The infection in the mother may be held in check sufficiently so as not to infect the fetus even though the treatment of the mother is entirely inadequate to bring about her cure. This is true whether or not the father receives treatment at the same time. In many instances a non-syphilitic child will result even if the mother receives treatment only in the later months of her pregnancy. No matter how late in her pregnancy the mother comes under observation, treatment is worth while, for even though the fetus is already infected, the infection will be modified by treatment and the child will have a better chance of life. Treatment of a mother during one pregnancy will have little or no effect upon a succeeding pregnancy unless the treatment of the mother is carried to the point of "cure." Both arsphenamin and mercury have been shown to be well borne in full doses by pregnant women. Such a mother is of course subject to reinfection unless her husband also receives adequate treatment.

In the treatment of syphilis there are three drugs in common use, *viz.*, arsphenamin products, mercury or one of its salts and potassium iodide. The manner of use of these drugs varies widely according to the opinions and experience of the clinician using them. For convenience they will be considered separately.

Arsphenamin and neoarsphenamin have proven sufficiently valuable so that every case of syphilis should be treated with one or the other. The weight of opinion is in favor of arsphenamin as the more valuable of the two, though no good evidence has been advanced that in sufficient dose neoarsphenamin is not just as effective. In children the ease of intravenous administration is much enhanced by the use of a simple syringe and needle. It is conceded by all observers that any dose of neoarsphenamin suitable for a child may be given with impunity in the small volume required for syringe administration. In the case of arsphenamin it is the opinion of many that a similar concentration somewhat exceeds the bounds of a reasonable caution, though by no means all agree on this point. Because of the greater ease of preparation and administration of neoarsphenamin this substance has many advocates. Both preparations have been used intramuscularly and by rectum. Intramuscularly they are very painful and sometimes cause sloughing. By rectum they are relatively ineffective unless given in much larger doses than is customary and suitable measures are taken for retention for a considerable period. Neoarsphenamin lends itself somewhat more readily to intrarectal administration than arsphenamin because the latter when suitably alkalized is much more irritating. Intravenous administration is the method of choice to such an extent that no other route can be recommended, when a vein can be entered. The cases in which some utilizable vein cannot be found without skin incision are so few as to be negligible. Any accessible vein answers. The veins at the elbow are preferable. The external jugular vein is usually easy to enter and is as safe as any other vein. In the case of infants, especially syphilitic infants, scalp veins are frequently large enough for use. In infants also the longitudinal sinus is easy of access through the fontanelle. This is mentioned last because it should be the last resort. The sinus is probably the easiest place of all to use for intravenous work in infants, but it is also the place of greatest danger should some slip or poor judgment allow part of the injection to escape through or around the sinus. Death may be caused very readily in this manner.

The dose of arsphenamin is 0.01 gm. per kg. of body-weight. This is slightly larger than is recommended at present for adults, but thousands of injections have shown it to be well tolerated. From 15 to 30 c.c. of freshly distilled, freshly sterilized water are used for each 0. 1 gm. The drug is first dissolved, then neutralized with sodium hydroxide and filtered. It is then ready for injection. It should be used very soon after preparation, since, in alkaline solution, it oxidizes readily to form toxic arsenoxides. When prepared in the dilution recommended, administration by the gravity method becomes necessary. The dilution recommended above is that advocated by the majority of sypholographers. In our clinic we have preferred arsphenamin to neoarsphenamin and at the same time have preferred the syringe method of administration. For convenience in syringe administration our dilution of arsphenamin has been 0.1 gm to 4 c.c. of water. In approximately ten thousand injections we have noted no ill effects which could be attributed to the concentration. Arsphenamin is purchased as the dihydrochloride which is quite acid and readily soluble. When this is just neutralized both molecules of hydrochloric acid are split off and the substance is insoluble. Further addition of sodium hydroxide forms the monosodium salt and still further addition the disodium salt of arsphenamin, both of which are soluble. Addition of alkali just to the point of solution of the neutral base produces the monosodium salt. The amount of sodium hydroxide required is 0.7 c.c. normal solution to each 0.1 gm. arsphenamin for the monosodium salt and 0.9 c.c. for the disodium salt. Though the monosodium salt is the one used by the majority of clinicians for injection, the disodium salt is claimed to give fewer reactions.

The dose of neoarsphenamin is 0.015 gm. per kg. of body-weight. For use the drug is simply dissolved in cold, freshly distilled, freshly sterilized water. No neutralization is required. Filtration is desirable. A safe dilution is 0.1 gm. to 2 c.c. of water, though by some a greater dilution is recommended.

The intervals between injections and the number of injections to a series varies with the experience and opinions of the clinician. A series of three to four injections at weekly intervals, repeated every two to three months has been shown to be effective. Injections daily or every two days for three doses have been shown to be safe if for any reason more intensive treatment is considered desirable. Certain exceptions and precautions may be noted. Very young and delicate infants with marked signs of active syphilis do not tolerate well the full doses frequently repeated at the beginning of their treatment. The reason that has been ascribed for this is that the killing of so many treponemata in so short a time results in a toxemia, sometimes fatal. Whatever the real reason, it is good practice to use small doses. Also in the case of involvement of the central nervous system, whether clinical or as shown by the cerebrospinal fluid, or in case the condition of the cerebrospinal fluid is unknown, it is good practice to precede arsphenamin administration with a course of mercury. The purpose is to avoid a Herxheimer reaction in the nervous system. The occurrence of a Herxheimer reaction in other parts of the body is not so important.

The use of arsphenamin is a great improvement upon the former treatment of syphilis with mercury and iodides but it has never fulfilled Ehrlich's hope that it would replace other drugs. To procure satisfactory and permanent results mercury must be used in addition to arsphenamin. Any preparation of mercury administered in any manner gives good results provided it is administered in such a manner as to be absorbed in sufficient amount and no damage is done to the patient. Mercury and its salts may be administered by inunction, by mouth or by intramuscular injection. Intravenous use is not advisable because of the irritating and sclerosing action upon the veins used. In dispensary practice pills are superior to inunction for the reason that these patients are much more likely to take the pills than to spend sufficient time with rubbing. In this same class injections of mercury salts are most satisfactory because one then is certain as to the regularity and dosage. The prevalent idea that intramuscular injections are not advisable for children does not seem to us to be well founded. Such injections have been a part of our routine for the past six years. A very satisfactory preparation for intramuscular injection is the bichloride. A suitable dose is 0.033 c.c. (0.5 minim) of a 1 per cent. solution per kg. (2.2 lb.) of body-weight, given twice a week, or given once a week and supplemented by daily mercury by mouth. One of the best preparations to give by mouth is metallic mercury finely divided in chalk, dispensed in tablet form and known as gray powder. The dose varies from 1/5 gr. twice daily for young infants to 2 gr. or more three times daily for larger children. Carthartic action is to be avoided. In the case of young infants, whose nutrition is precarious, mercury by mouth is often less suitable than other methods of administration because of the cathartic action it may have Mercury by inunction is best given as metallic mercury finely divided in an ointment base. The official preparation is known as blue or mercurial ointment. The dose varies from 1 gm. (15 gr.) for young infants to 4 gm. (1 dr.) for larger children. This amount should be well rubbed into the skin once daily, different parts of the body being chosen to avoid irritation. In delicate infants the ointment may be spread to advantage upon the inner surface of an abdominal binder. Considerable absorption is obtained without rubbing. Mercury administered to a mother is excreted in appreciable amounts in the breast milk. However, this method of administering mercury to an infant is inadequate and unreliable.

The arsphenamin and mercurial treatment should continue until the patient is in the condition which, according to our present standards, is considered as "cured." However, it is advisable for a number of reasons to have from one to three rest periods of a month to six weeks each during each year of treatment. The criteria of cure are discussed in another place. A period of intensive mercurialization commonly causes a slight albuminuria. In most instances this may be disregarded since the albumen promptly disappears during the rest period and no permanent abnormality results. It is desirable at all times and especially necessary in the case of infants to observe the nutrition of the patient. An intensive anti-syphilitic regime may cause stationary or loss of weight and in such a case the treatment should be diminished or stopped altogether for a time. In many instances it may be advisable to administer a tonic during the rest periods.

The importance of breast feeding of infants is pointed out in another chapter. It should be emphasized that maternal nursing or at least a human milk diet is more of a necessity for the syphilitic infant than for one who is not.

The iodides have no effect upon treponemata but are useful in aiding absorption of exudative and gummatous processes. Consequently they are much less useful in infants than in older children. The pain of syphilitic periostitis is very promptly relieved by iodides. Either sodium or potassium iodide may be used. A suitable dose is 0.2 to 0.5 gm. (3 to 7.5 gr.) three times daily according to the size of the child. Larger doses are unnecessary and serve no useful purpose.

The treatment outlined above serves for the general management of syphilis. It is often desirable, however, to direct special attention to certain local processes. The fissures resulting from infiltration of the skin about the mouth may improve somewhat more rapidly if treatment with a 5 per cent. silver nitrate solution. The pain of epiphysitis may be relieved by fixation of the part until anti-syphilitic treatment is effective. The syphilitic rhinitis of infancy makes nursing difficult and sometimes almost impossible. Anti-syphilitic treatment is usually slow to give relief. The dropping into the nose of liquid petrolatum or a 0.2 per cent. solution of menthol in liquid petrolatum at frequent intervals usually affords considerable relief. Also the application into the nose of 1 per cent. yellow oxide of mercury ointment is of benefit. Condylomata may be treated by the local application of powdered calomel. Late neglected bone syphilis may break down and result in a sinus through which secondary infection is likely to occur. Surgical interference as for chronic osteomyelitis then becomes necessary in order to secure healing.

When, either by clinical observation or by examination of the cerebrospinal fluid, the central nervous system is known to be involved it is found that in most early cases and in many late cases the general systemic treatment as outlined above is effectual in accomplishing at least a serologic cure and frequently a clinical cure. There can be no replacement of destroyed nervous tissue. If after six months to a year of such treatment no improvement is noted either in the clinical manifestations or the pathology of the cerebrospinal fluid it then becomes desirable to administer treatment intraspinally. Though the value of intraspinal therapy is in dispute the weight of evidence is in favor of the usefulness of this measure. Our personal experience would also bear this out. The injection of either neoarsphenamin or arsphenamin in aqueous solution directly into the subarachnoid space has been shown to be dangerous because of the irritant effects. Serum arsphenaminized either in vitro or in vivo is much less irritant and quite safe in proper dosage. Serum obtained from blood withdrawn ½ hour after intravenous arsphenamin contains in the neighborhood of 0.023 mg. of arsphenamin per cubic centimeter. The amount of serum injected depends upon the amount of cerebrospinal fluid withdrawn and varies from 10 to 25 or 30 c.c. according to the size of the child. Inactivation of the serum at 56°C. for ½ hour increases its efficacy. (The original Swift-Ellis technique uses serum from blood withdrawn one hour after arsphenamin.) There are those who further fortify such a serum with arsphenamin and still others use the Ogilvie technique of adding all of the intended dose of arsphenamin in vitro. Mercurialized serum may also be used for intraspinal treatment according to the method of Byrnes. Our own routine at present consists of the injection of serum arsphenaminized in vivo as described above with the addition of 1 to 2 mg. of mercuric chlorid before inactivation. The intraspinous treatments are more effective when combined with intravenous. They may be repeated every 5 to 10 days depending upon the amount of reaction from the treatment. Three to five such injections may be made to a series.

Reactions after Arsphenamin.—The intravenous administration of arsphenamin may be followed by various unpleasant or even serious reactions even though carried out according to a standard technique in which due attention is given to the purity of the water, the temperature, concentration, neutralization and rapidity of administration of the solution. The most frequent of these reactions is that which comes immediately after or during the administration, and is sometimes spoken of as a nitritoid reaction. Marked redness of the face occurs, sometimes succeeded by a cyanotic color. In case of severe reactions there is swelling of the lips, tongue and eyelids, injection of the conjunctivæ and lachrymation and an anxious expression of the countenance. Nausea, vomiting and retching followed by profuse perspiration is frequent. There may be a feeling of suffocation or air hunger with cough, respiratory embarrassment and dyspnea. The pulse is at first full and bounding, later slow and of small volume associated with pallor. These symptoms subside spontaneously in 15 to 30 minutes. Their cause is still in dispute but there is evidence that they are the result of circulatory damage brought about by the drug itself or by an impurity associated with it. This does not furnish an entirely adequate explanation because some children exhibit some or all of these symptoms with great regularity while others never show them even though given the same solution. The hypodermic or intramuscular administration of epinephrin will prevent or abort the attack.

Less commonly another group of symptoms may appear within a few hours of the administration and last from 12 to 24 hours or more. These may consist of chilliness or rigor, headache, vertigo, nausea, vomiting, diarrhea and a slight fever.

None of the symptoms of reaction mentioned above are as a rule serious or the condition of the patient alarming, but a third group occurs, fortunately rarely, which is serious and frequently fatal. These are referable to the brain, the liver or the skin. In several thousand arsphenamin injections in children we have observed but one reaction of each type, and but one of them (encephalitis) failed to recover. All three of these reactions followed the administration of neoarsphenamin. The most serious and uniformly fatal of these reactions is referable to the brain. All apparently goes well until the third or fourth day when there develops suddenly and with a rapid course headache, vomiting, extreme agitation, muscular twitching, epileptiform convulsions, dilatation of the pupils, absent reflexes, coma and death 24 to 48 hours after the onset. Coma is associated with facial congestion and fever. At necropsy disseminated punctiform hemorrhages are found throughout the brain. The brain cells are normal and marked edema separates the nerve fibers. Milian has saved such a patient by means of intravenous epinephrin while others have failed with the same therapy.

Severe jaundice accompanied as a rule by fever may have its onset from three days to several weeks after arsphenamin injection. Most cases run a favorable course but exceptionally death results with the symptoms and findings of acute yellow atrophy. The one patient we have seen with this disorder had several arsphenamin treatments before the jaundice appeared and numerous injections since with no further symptoms referable to the liver but with a fairly marked immediate (nitritoid) reaction after each injection.

Persistent eruptions on the skin may be observed following arsphenamin. Apparently the only serious occurrence in this connection is a universal exfoliating dermatitis which persists for weeks with fever and debility and sometimes with a fatal result.

A Herxheimer reaction following arsphenamin differs from those described above in that it appears within 24 to 36 hours after injection and consists of an aggravation of symptoms or lesions already present. This reaction is not of frequent occurrence. Ehrlich ascribed it to inadequate action of the drug which serves to irritate and not to kill. Whether it is due to this or the freeing of toxin from the death of treponemata, or a vasotropic action of the drug or some other reason is unknown.

CRITERIA OF CURE

In discussing the subject of what constitutes a cure in hereditary syphilis the nature of the disease must be kept clearly in mind. Whether or not one can consider a syphilitic child as cured depends entirely upon the definition of what constitues a cure. In so far as we can judge it is possible to produce a serological cure or negative reaction which persists, which we interpret as meaning the death of all syphilitic organisms or the production of a state in which they are persistently inactive. That is to say the specific infection has been overcome. On the other hand the morphological changes produced during the active period of the infection may persist and although there may be no further progress a blind or a mentally deficient child may be the permanent result of the infection.

It is the common impression that hereditary syphilis is cured with difficulty or not at all. There may be some question as to the absolute cure of the disease, but with the modern methods of treatment a persistently negative Wassermann reaction and freedom from symptoms has been the rule in our hands. Before declaring a patient as possibly cured Fournier required a minimum of three to four years of treatment and a period of absence of symptoms for 18 months to two years. Modern arsenical treatment has permitted the shortening of the treatment period. It is also recognized that an infection treated early is more quickly eradicated than one treated late, so that much depends upon the stage of the disease. Even with our more modern methods it is scarcely safe to treat an infant less than one year or an older child less than two years regardless of the effect treatment has had upon the Wassermann reaction. If either the blood or cerebrospinal fluid Wassermann reaction is positive after this time treatment must be continued until both are negative. In some cases as much as three years or more of treatment may be required. Observations should then be made at intervals for several years both in regard to the Wassermann reaction and recurrence of clinical manifestations. Should there be a recurrence the treatment should be undertaken as if none had ever been given. To the present our experience has been that once a spinal fluid is negative it always remains negative. When a spinal fluid is once known to be negative lumbar puncture need be made again only for final discharge. Amounts of spinal fluid up to 2 c.c. should be used for the Wassermann reaction and in addition there should exist no other pathology such as positive colloidal gold, globulin, or increased cells. When determining the status of the infection by means of the Wassermann reaction all treatment should be discontinued for at least two weeks prior to taking the blood specimen.

ACQUIRED SYPHILIS IN CHILDHOOD

Acquired syphilis in childhood is more a subject of historical than practical interest. If one turns to the older pediatricians or syphilographers extensive discussions of infection acquired from wet nurses will be found, and we have every reason to believe that this method of infection was not at all uncommon. The almost complete disappearance of the wet nurse in America, together with the modern diagnostic methods available for examination of the few wet nurses employed, has practically wiped out this method of infection. In the past 10 years we have not encountered a single instance in the material of the St. Louis Children's Hospital and affiliated clinics of infection acquired in this manner. No woman should ever be employed as a wet nurse until after the blood has been tested and the Wassermann reaction found negative.

Acquired infection, when it does take place, may be extra genital or genital. In some 30 odd cases of extra genital chancre observed in the clinics associated with the Medical School in recent years, but two were in children under 14. Both of these were lip chancres from kissing.

Other methods of infection and extra genital location of the chancre are in the form of medical curiosities.

Acquired syphilis in children is far more apt to be genital in origin and a number of such cases have come under our observation. In every case they have followed a criminal assault or the practice of some form of perversion. The infection in these cases follows the course of acquired infection in the adult and goes through the regular primary and subsequent stages, and there is practically nothing in the diagnosis or treatment which requires special mention. Condylomata about the vagina or anus have been quite extensive in our cases and this we have attributed to the marked trauma which is usually associated with the assault. Treatment is by arsphenamin and mercury and the removal of the child after it becomes non-infectious to a new social environment. In one of our cases the original infection in a boy of 10 years was cured by intensive and continued treatment only to be followed later by a second initial lesion as the result of renewed perversions. The importance attached to the subject of acquired syphilis in infancy and childhood by earlier writers no longer exists.

CHAPTER CXXIV

ERYTHEMA INFECTIOSUM

By Henry L. K. Shaw, M.D.

Albany, N. Y.

History.—Erythema infectiosum is a feebly contagious disease occurring chiefly in children, with very slight subjective symptoms, characterized by a maculopapular rose-red rash, which is more pronounced on the cheeks, legs, and outer surface of the arms. The exanthem was first recognized by Escherich in Graz, during an epidemic which accompanied an outbreak of measles.

Tschamer, in 1886, gave a very accurate description of the disease under the name of "Oertliche Rötheln." He recognized in this condition a very different type of eruption from rötheln, but came to the conclusion that his cases were an abortive form of German measles. This epidemic which gave Tschamer an opportunity of observing this disease was described by Gumplowiez in 1891 under the title, "Casuistisches und Historisches Über Rötheln," in which he agrees with Tschamer in its being an abortive type of rötheln. He showed that measles conferred no immunity over this disease and *vice versa.*

In a paper written in 1896 entitled, "Zur Polymorphie und Differentiale Diagnose der Rubella," Tobeitz confirmed the observations and conclusions of Tschamer. He reported a number of similar cases which showed "a distinct departure from the general course of rubella." He claimed this polymorphism was characteristic of rötheln, and was produced by some change in the biology of the infecting organism effected by the season, surroundings, or some condition or idiosyncrasy of the individual. Escherich made the first claim that this form of eruption, instead of being identical with rötheln, was a disease sui generis. He had a further chance to study this disease during two epidemics in Graz in 1897 and 1899. In the same year one of his assistants named Schmid, after observing 121 cases, gave a full description of the disease and its essential characteristics.

In 1899, Stricker of Giessen writing in a local medical journal gave an account of an epidemic of "Die neue Kinderseuche in der Umgebung von Giessen, erythema infectiosum." He was the first to give the disease this name which was accepted and adopted by Escherich. A full description of 45 cases was given by one of Stricker's students in an inaugural dissertation.

Tripker, a pediatrist in Coblenz, described in 1901 an epidemic of 70 cases under the title, "Erythema Infectiosum febrile oder epidemischer Kinderrothlauf." An analysis of his cases shows them to be identical with erythema infectiosum, although some were accompanied by a slight fever. An epidemic occurring in Berlin in 1901 in the same families and in about the same locality was described by Feilchenfeld under the name "Erythema simplex marginatum."

Plachte, a dermatologist, observed an epidemic of this same exanthem which he described in 1904 as "Megalerythema epidemicum die Grossflecken, ein neues Kinderexanthem." He did not think the term erythema infectiosum was descriptive of the disease, as it could be equally applied to scarlet fever or measles.

Heimann reported before a meeting of Niederrheinische Kinderärzte at Cologne, an epidemic of this disease in Solingen in 1903, and which he termed erythema infectiosum morbilliforme, on account of the similarity of the exanthem with measles.

Pospischill, the attending physician to the contagious pavilions of the Wilhelmina Children's Hospital, made a preliminary report in 1904 on "Ein neues als selbstandigerkanntes akute, Exanthem." He gave a clear and unmistakable description of erythema infectiosum, which was not infrequent in Vienna, and differentiated it definitely from measles, scarlet fever, and German measles. Patients have been sent to him with a diagnosis of measles or even scarlet fever.

The complete report of these cases appeared in an article in the Wiener klinische Wochenschrift for June, 1904, in which Pospischill states that the cases formerly described by him as scarlatinoid and morbilloid were in reality erythema infectiosum.

Weber in 1916 reported a rather extensive epidemic in Switzerland and he was convinced of the clinical entity and infectiousness of the disease. Tobler in the Berliner Klinische Wochenschrift, No. 12, 1914 reported an epidemic of 38 cases in Breslau. A description of an epidemic of this disease is given by Ochsenius in the Muenchner med. Woch., page 830, 1917 and is the most recent German reference. The description of erythema infectiosum has appeared chiefly in the German literature. As yet, this disease as such has scarcely been recognized in America.

A personal communication from Doctor Braner of Hamburg, N. Y., gives an account of an epidemic of 37 cases which occurred in that town in the spring of 1919. As this is the only report of an epidemic in this country the following abstract from Doctor Braner's letter will be of interest.

"As per your request I am giving you a few details of our epidemic of erythema. I have had an opportunity to study 37 cases, 24 females and 13 males. Two of the female cases were adults.

"The course of the disease is usually as follows:

"Without prodromes the eruption appears first on the face and the patient presents the appearance of being over heated but the color is a dusky-red, especially on the cheeks the color showing a well-marked line of demarcation and on close inspection is seen to be composed of numerous macules. The skin feels very hot to the touch and the patient complains of a burning sensation.

"On the following day the eruption makes its appearance on the extensor surfaces of the arms and shoulders moving down to the forearms, this eruption being maculopapular and resembling, in the majority of cases a measles rash. Usually on the second or third day a macular eruption is seen on the trunk—slightly on the chest—the chest sometimes being entirely free from any eruption. The maculopapular eruption is marked on the buttocks where it may become confluent. Next the same type of eruption appears on the anterior surfaces of the thighs and legs.

"As the eruption fades it leaves on the arms, legs, shoulders and buttocks annular and gyrate figures or a map-like net work which I have seen as late as three weeks after the initial rash. There is also a peculiar marble-like mottling of the skin which is quite evident at times after the rash has faded. There is one constant and characteristic feature of this eruption and that is its evanescence. It may appear to a marked degree in certain locations while one is making an examination. It also disappears very suddenly only to reappear in a few hours or days.

"There is one feature of this disease which I have not seen mentioned and that is an evanescent vermillion coloring of the tip and edges of the tongue. It appears and disappears while making an examination.

"I have never seen any eruption on the fingers.

"A striking feature of the disease, is the complete absence of symptoms in most cases, although some begin with a slight fever, restlessness, coryza or even slight angina.

"I have seen different types of this disease. In young infants it is a typical measles eruption minus Koplik spots. In one case I saw a discrete punctate rash which resembled somewhat that of scarlet fever, the chest being almost free from eruption. Oddly enough the first case I saw was a hemorrhagic type. One case very closely resembled facial erysipelas.

"I have not found albumin in a single case although a few showed indicanuria.

"As a rule there is no desquamation although I saw in one case a furfuraceous desquamation and in the case resembling erysipelas a localized desquamation of the face.

"The incubation period is apparently 10 days to three weeks."

Doctors Wende and Snow of Buffalo have been in consultation with me and have studied a number of the cases. They are convinced that this disease is distinct from scarlatina, measles and rubella and corresponds in every detail with the description and course of Erythema Infectiosum."

The so-called "Fourth Disease," described by Dukes in 1900, differs from it in all essential respects, and will be considered later in discussing the differential diagnosis.

Etiology.—The specific agent is unknown. The disease occurs in epidemics, and often follows an outbreak of measles or rötheln. It is found to spread through families, while a number of cases have been observed occurring simultaneously in schools and kindergartens. Although it is undoubtedly carried by contagion, it is not so infectious as the other exanthemata. Patients have been admitted to the wards of the hospital, in Vienna and Graz before the nature of the disease was recognized and no outbreak of measles or rötheln has resulted. An attack of the other exanthemata affords no immunity against erythema infectiosum.

The age at which children are most commonly affected is between 4 and 12 years. The youngest recorded case was in an infant, aged 14 months; several cases have been observed in young adults. It is essentially, however, a disease of early childhood which affects both sexes in about equal proportion. It occurs most frequently in the spring and summer months.

The specific agent, mode of transmission and life of contagion are unknown. As the disease is not found frequently it may be assumed that contagiousness is very limited or the predisposition not general. A number of Weber's cases occurred in an orphan asylum and only two or three cases developed each week.

Symptoms.—In cases where it was possible to make correct observations, the period of incubation was from 6 to 14 days. The disease may be ushered in by a slight feeling of malaise, weakness, and sore throat, but in the majority of cases, the first symptom noticed is the eruption. This is the most important and often the only symptom. It appears invariably on the skin, and causes no constant changes on the mucous membranes. A diagnostic feature of the disease is the character of the rash on the face, where it first makes its appearance. The cheeks which are the parts chiefly affected present symmetrical rose-red efflorescence. The skin is swollen, hot to the touch, but is neither sensitive nor itching. The color disappears on pressure, but quickly reappears. The whole appearance is suggestive of erysipelas. The eruption is confluent over the cheeks. The edges are well-defined, slightly raised, and usually distinct from the normal skin, but they may gradually fade into it. The area of confluent eruption is rather sharply limited in front by the nasolabial folds making the skin around the mouth appear pale in contrast to the livid hue of the cheeks, while above, it is limited to the temples. Laterally, the eruption extends to the angles of the jaws. Discrete spots varying from the size of a pea to a hazelnut, are often seen on the forehead and chin. The rash fades from the face after four or five days. On about the second day, the eruption makes its appearance on the body. It is most marked on the outer surfaces of the arms and legs. The trunk is involved to a much less degree, and may be almost free, but in no case is the rash so extensive as on the face and extremities. The eruption spreads toward the periphery, affecting the hands and feet last. On the extremities, the exanthem is typical and characteristic. It is morbilliform in appearance, and not so deeply rose-red as on the face. The contour of the eruption frequently presents almost geographical outlines. In many cases, the appearance is suggestive of lace work, especially at the end of the disease when the eruption begins to fade. On the inner or flexor surface of the arms, the eruption is not nearly so intense, whereas around the outer surface of the elbow it is apt to become confluent. On the legs, the eruption is similar to that on the arms. It is always especially well marked over the buttocks. The trunk remains comparatively free from eruption, although a number of discrete spots, sometimes crescentic in form, can be seen sparsely scattered over the chest and back.

The rash is more macular than papular, and shows only a slight elevation, except on the face, where it is always raised. An evanescence peculiar to this disease is often observed. The rash will disappear, and then some slight irritation to the skin, such as friction, exposure to cold, will bring it out again in full bloom. The eruption lasts from 6 to 10 days. It is not followed by desquamation and does not leave any stains or markings, such as are sometimes seen after measles. No hemorrhage results on pinching the skin, as occurs in measles and scarlet fever. The lymphatic glands are not enlarged as a result or accompaniment of this disease.

The subjective symptoms are conspicuous by their absence. The tongue may be slightly coated, but it never presents the strawberry appearance of scarlet fever. The conjunctivæ are not congested, and there is no coryza or cough. The urine is normal.

Differential Diagnosis.—The disease most likely to be mistaken for erythema infectiosum is rötheln. In this disease however, prodromal symptoms are not uncommon. The rash spreads rapidly over the entire body, especially over the chest and back, appearing first as small, discrete spots, somewhat larger than the punctate spots of scarlet fever, and smaller than the papular rash of measles. On the face the favorite locations of the rash are the region around the mouth, back of the ears and on the forehead. The color of the eruption is pale pink. The lymph-nodes, especially those of the post-cervical group, are invariably enlarged, and while there may be only a few constitutional symptoms, yet a temperature of 103°F. is not uncommon. In erythema infectiosum, the appearance, location and spread of the rash is entirely different. The lymph-nodes are not enlarged, while fever occurred in only a small percentage of the reported cases. During all three of the epidemics in Graz, erythema infectiosum, was often seen in children known to have had measles or rötheln. As it accompanied or followed outbreaks of these diseases, the difference was easy to establish. As it not infrequently is associated with these diseases, the importance of the differential diagnosis is all the greater.

Measles can be excluded by the absence of all involvement of the mucous membranes, of all constitutional symptoms, and the uniform absence of the Koplik spots. Although the appearance of the rash on the arms and legs is strikingly similar to measles, and may cause a momentary confusion, a further examination will clear up the diagnosis. The question of its being a second eruption of measles with atypical symptoms has been raised. This is not tenable, because of the fact that erythema infectiosum very often occurs in children who have never had an attack of measles. It is more probable that the so-called recurrent attacks of measles are in reality, cases of erythema infectiosum.

Scarlet fever should present no difficulties, although this diagnosis has more than once been made in cases of erythema infectiosum. The throat symptoms, the color and location of the rash, the constitutional symptoms, have nothing in common with erythema infectiosum.

Erythema exudatum multiforme, which was first described by Hebra, presents more similarity in name than in clinical features. Here the back of the hands and feet are first affected; while in erythema infectiosum, the face is the first and most characteristic seat of the eruption. In the Hebra type, the rash is first macular, then papular, and later vesicular. The disease lasts for three or four weeks, and is generally accompanied by severe constitutional symptoms. It is never contagious and can frequently recur in the same person.

Pityriasis rosea presents some similarities in the skin lesion, but it seldom affects the face, is never contagious, and runs a chronic course of from 8 to 10 weeks.

Drug rashes, toxic and dyspeptic erythema, can be differentiated by the history. At first sight these rashes, especially the serum and toxic erythema, are very similar to the rash of erythema infectiosum. The specific cause, the duration, location, and course of eruption, should remove any possibility of confusion. The same can be said in regard to urticaria where the further absence of itching and of characteristic wheal formation makes the diagnosis clear.

Very little has been heard of the "Fourth Disease" in the past two years, and the medical profession seems to have accepted Ker's verdict of "not proven." Any description of a new exanthem will at once recall the article of Clement Dukes, which appeared in 1900, entitled "On the Confusion of Two Different Diseases under the Name of Rubella (Rose-rash)." The rash, which is the most characteristic feature of both diseases is very dissimilar. In the fourth disease it is very extensive and covers the entire body within a few hours. Its color is bright-red, and from the character of the rash alone, "it is impossible to distinguish it from scarlet fever." The face often remains free from any signs of eruption. Profuse desquamation follows in a number of cases. Without entering into a further discussion of this disease, it will be plainly seen that the essential characteristics of each disease are different. Ker, of the Edinburgh City Hospital for Infectious Diseases, in discussing the question, "Is there a fourth disease?" said that he had long been prepared to hear that there was a new disease hitherto confounded with rötheln, but he could not be convinced that the fourth disease of Dukes' met all the requirements.

Treatment.—There is no specific treatment for this disease. The constitutional symptoms are so slight that very little treatment is required. As the period of contagiousness is unknown it would be wise to keep the patient isolated until the eruption has entirely disappeared. A rigid quarantine is not indicated.

The patient should be kept in bed during the eruptive period. The rash itself does not give rise to any subjective symptoms and it will not be necessary to use any lotions or ointments.

CHAPTER CXXV

ERYTHEMA NODOSUM

By Henry L. K. Shaw, M.D.

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Erythema nodosum is an acute infectious disease which presents a peculiar bruise-like eruption on the skin and is characterized by a period of incubation, of invasion, of eruption, and of desquamation. It is mildly contagious, and like the exanthemata, finds its greatest number of victims among the young. The disease has been variously regarded as the cutaneous expression of a systemic disorder due to absorption of toxins; as an evidence of syphilis or tuberculosis; and as a distinct disease due to a specific microörganism. The term erythema nodosum was first applied to this condition in 1799 by the French physician, Robert Willan, who described the disease in his textbook on diseases of the skin.

Pathology.—The typical lesions are found on the skin and consist of a collection of inflammatory areas of varying size. There is an exudation into the dermis and hypodermis, the exciting cause being undoubtedly a toxin. The presence of some degree of angioneurosis seems to be a factor in the process. Hemorrhage, infiltration, and edema surround a blood-vessel, and hence its adjoining nerve, thus occasioning much local pain. A nodule of fibrous nature freely movable under the skin was reported by one observer.

Etiology.—The specific organism has not yet been isolated, but considerable evidence has been collected to prove its contagiousness. It is essentially a disease of childhood, affecting both boys and girls about equally. In adult life, however, the disease is very rare among males, but still quite common among females. Seasonal incidence of erythema nodosum shows a greater prevalence in spring, summer, and early autumn. The cases are usually sporadic, but occasionally two or more persons of the same family are affected at the same time. The disease is rare in infancy, most cases occurring in children over three years of age. E. Fuhrmann reported a case in a newly born infant. Statistics bear evidence that the maximum of incidence is reached about the tenth year of life.

Anemic, debilitated, or scrofulous children are particularly liable, though perfectly normal individuals are likewise attacked (Abt). It may occur either in the midst of one of the infectious diseases or following an attack of rheumatism. The long existing uncertainty as to the contagious nature of the disease led to numerous observations along this line. Moussons observed that a child who was admitted to a hospital ward with erythema nodosum seemed to infect another child in an adjoining bed. Heim reported that a child who fell sick with erythema nodosum seemed to communicate the disease to a little sister in whom the usual symptoms developed.

Rosenow, as the result of recent experimental study on the etiology of this disease, concludes that a diphtheroid bacillus, closely resembling the streptococcus group, is the causative agent. He produced the disease in guinea pigs and rabbits by intravenous inoculation of the organisms isolated from the blood and excised nodes. He believes the tonsils and pus pockets about the teeth play a part in lodging and disseminating the infection.

Association with Tuberculosis.—Many writers are of the opinion that this disease is the concomitant of a tuberculous process. Landouzy has presented experimental evidence which shows that tubercle bacilli were present in the blood and skin lesions in some cases, a fact which has been accepted by some as conclusive evidence of the tuberculous etiology of this disease. Pollak found a strongly positive von Pirquet reaction in all of the 48 cases of erythema nodosum in children between 2 and 13 years to whom he applied the test. On the basis of these findings he states that in childhood the disease occurs exclusively among individuals infected with tuberculosis and is in all probability a tuberculous dermatosis. Three of these children later developed an active tuberculosis of the cervical lymph-nodes, meninges, and pleura; while several had a history of antecedent tuberculosis of glands and bone.

Brian, after careful experimental study, concluded that tuberculosis is certainly not the only cause of erythema nodosum, and probably enters into the etiology of only a small number of cases. Hildebrandt believes in the tuberculous origin of some cases, but maintains that final proof is still lacking. Kober is of the opinion that erythema nodosum occurring in children with a family history of tuberculosis should be regarded as an early symptom of infection with the tubercle bacillus, and an indication for prophylactic measures.

Various French observers, notably Landouzy, Aubert, Alamartine, and Leriche, believe this disease is a septicemia due to the tubercle bacillus; while Courmont and Pic believe many cases to be non-tuberculous. Sézary states that tuberculous meningitis is a common sequel of erythema nodosum. Kuhn obtained a history of tuberculosis in 8 out of 22 cases, though he considers the association of the two diseases as a coincidence. The literature contains the reports of many cases occurring in children in whom tuberculous meningitis or other forms of tuberculosis later appeared.

Stokes of the Mayo Clinic in the American Journal of Medical Sciences, February, 1919, emphasizes the tuberculous origin of this disease.

Association with Rheumatism.—So frequently are the two diseases associated with each other that many have regarded erythema nodosum as a manifestation of rheumatism. As Stephen McKenzie has shown, it is frequently associated with definite rheumatic symptoms, such as articular pains, sore throat, and acid sweats, and it may be complicated by endocarditis without the existence of articular lesions. Nevertheless, in certain features, the two conditions show striking differences. Rheumatism occurs mainly in November and is least common in March; while the incidence of erythema nodosum is highest in March and low in November, showing no relation between seasonal incidence. Rheumatism occurs in both sexes in about equal proportion; erythema nodosum is three to five times more frequent in females, and is peculiar in that the incidence in the male sex ceases almost entirely at puberty. Again, in rheumatism recurrence is frequent, while in erythema nodosum, history of a previous attack is never obtained. Rheumatic symptoms, particularly the pyrexia, yield readily to salicylates, which have no influence on the pyrexia of erythema nodosum.

Symptoms.—The incubation period covers an interval of about 8 to 10 days. The invasion occupies about three days to a week, during which, as a rule, the symptoms are slight. Malaise, anorexia, and some gastric disturbance are present. In some cases there is an irregular temperature, some prostration, and more marked gastric disturbances. More rarely, the condition may simulate typhoid fever or even suggest meningitis. The onset is frequently marked by an attack of tonsillitis which may subside and be followed for a few days by an absence of all symptoms until the general infection manifests itself.

The attack may begin with shivering or even a rigor, but more often the onset is insidious and characterized by headache, vomiting, and anorexia. In severe cases, pain in the back is very frequent; joint pains and even effusions may occur in the knees, ankles, or wrists. Conjunctivitis has been observed and occasionally a phlyctenule. There is some pyrexia in the prodromal period, and the sweating is often profuse.

Occasionally, no preliminary warnings precede the rash. As a rule, however, the foregoing train of symptoms is present for a variable length of time up to two weeks before the characteristic nodes appear. These make their appearance earliest and most abundantly on the legs, especially on the anterior and inner aspects, and frequently on the arms and forearms, hands and feet, and even on the face and trunk. The lesions are frequently raised above the surrounding skin surface, the elevations often being visible, but readily determined by touch. They vary much in size, but are commonly ½ to 1 in. in diameter. They are smooth, indurated, and at times fluctuating. The nodes have no well-defined border, are round or oval in shape with the long axis corresponding to the axis of the limb They do not itch, nor do they tend to suppurate, but are painful, both subjectively and to the touch. Their color is bright-red, and somewhat glossy, as if polished. As the lesion grows older, the color takes on the changes peculiar to a bruise. So characteristic is this, that the French use the synonym *dermatite contusiforme.* The nodes occur in crops, each lasting a few days, so that the lesions are seen in various stages of development and retrogression. They fade in a period of a few days to three or four weeks, and as they disappear, a fine desquamation may occur.

At this stage, the tongue is usually thickly coated. The pyrexia often persists for a few days after the appearance of the rash; sometimes it is septic in type; more often it does not rise above 101 or 102 degrees, and it drops, as a rule, within a week to normal. Until this stage, there is frequently more or less cardiac disorder; often a soft systolic murmur at the apex and an increase in heart dulness. Occasionally albuminuria occurs. Severe anemia usually results from the infection.

Diagnosis.—The disease is recognized by the typical appearance of the skin. The diagnostic feature is the development, in connection with a moderate febrile disturbance, of multiple painful nodes in the skin, showing a predilection for the legs; the nodes become boggy show no tendency to suppurate, and undergo the involution of a bruise.

Differential Diagnosis.—The connection of erythema nodosum with tuberculosis and rheumatism has already been considered. The other diseases likely to be confused with it are syphilis, erythema multiforme, and areas of cellulitis.

In **syphilis** the lesions develop much more slowly and break down. They are promptly affected by potassium iodide, which has no effect on those of erythema nodosum. In congenital syphilis the lesions may also be multiple, but other definite evidences of the disease should be present. The Wassermann reaction would aid in the differentiation of the two conditions.

Erythema multiforme presents the appearance of being merely a local eruption and lacks the characteristics of an exanthem with a period of incubation, of invasion, of eruption, and of desquamation, and signs of constitutional disturbance. In erythema nodosum, the lesions are more painful, inflammatory in character and are deeper seated.

Areas of cellulitis are more diffuse and usually larger, and tend to suppurate.

Prognosis and Course.—Erythema nodosum, *per se,* is not a fatal disease. The average case runs its course in two to three weeks time, but convalescence is often slow and protracted, owing to the anemia which so frequently results from the disease. There is not likely to be a recurrence, but permanent damage may be left in its wake, and the possibility of a resulting endocarditis should be borne in mind.

Complications and Sequelæ.—Cardiac disease and tuberculosis deserve first place on this list. Rarer complications have been noted by various observers. Hemorrhagic nephritis was observed by

Curschmann; various neuralgias by Lewin; bilateral paralysis of the perineal nerves by Quincke; multiple neuritis by Baumner; and psychoses in the form of mania and melancholia by Jolley.

Treatment.—Because of the contagious nature of the disease, the patient should be isolated and proper prophylactic and quarantine measures put into effect. Rest in bed is essential both for easing the pain in the limbs and as a quietus to the inflammatory process. For further relief of the pain, it is well to apply lead and opium wash to the nodes and to bandage the legs firmly over a thin layer of absorbent cotton. Ten per cent. ichthyol ointment is a valuable application. Jamieson advises painting the nodes with flexible collodion

The bowels should be kept open with saline cathartics. Internal administration of salicylates and quinine is believed to be helpful.

During the acute febrile period the diet must be limited to liquids and soft foods. During convalescence the heart should be carefully watched, and the child kept quiet until its function is properly restored. Iron and arsenic are indicated to combat anemia.

CHAPTER CXXVI

BUBONIC PLAGUE

By Victor G. Heiser, M.D.

New York City

Bacteriology.—The specific organism of plague was discovered by Yersin and Kitasato,[1] working independently in Hongkong in 1894. The Bacillus pestis may be found in the blood, acutely enlarged lymph glands, the sputum, the organs, especially the spleen, and elsewhere in the human body. The organism may usually be recovered in pure culture from characteristic buboes. It is short, thick coccobacillus, 1½ to 2 microns in length and from 0.5 to 0.7 micron in breadth. It has round ends and resembles the organism of chicken cholera. It is a bipolar staining bacillus and is usually decolorized with the Gram method. Films can be made of fluid drawn from the suspected bubo and stained with methylene blue which has been diluted with carbol-fuchsin.

Etiology.—Plague is caused by the Bacillus pestis. Typical attacks are produced when the organism is inoculated into monkeys, cats, rats, guinea pigs, squirrels, mongooses, bats, tarabagans, and marmots. In bovines and equines it only produces local reactions. Canines, birds, and reptiles are apparently immune. Plague causes epizootic among rats, either in acute or chronic form. In the latter condition it probably maintains itself between epidemics. The true reservoir of the disease is the rat, and to a lesser extent the squirrel and other rodents; man is only its accidental victim. The Indian Plague Commission[2] of 1908 came to the following conclusions: "Contagion occurs in less than 3 per cent. of the cases, playing a very small part in the general spread of the disease. Bubonic plague in man is entirely dependent on the disease in the rat. Infection is conveyed from rat to rat and from rat to man solely by the means of rat fleas. A case in man is not in itself infectious."

Pathogenesis.—The great majority of human plague cases originate from flea bites, the inciting fleas having been infected by feeding upon rats suffering with the septicemic type of the disease. The latest researches indicate that the plague organisms regurgitated by the flea are rubbed into the wound produced by the bite. At rare intervals it is possible to find the probable site of the entrance infection, and plague bacilli may be recovered from the skin and underlying tissues involved in the wound produced by the bite. The bacilli, after being introduced, travel by way of the lymphatics to the nearest lymphatic glands. The resisting power of the glands is almost invariably overcome, and the infection then spreads via the thoracic duct to the blood stream, and causes septicemia. Another mode of entrance into the blood is probably through the direct absorption caused by the degeneration of the cells in the lymphatic glands. The glands most usually affected are the femoral, the inguinal, the axillary, and the cervical. It is noteworthy, however, that while most cases of plague first show enlargement of the glands of the groin, it does not necessarily mean that the infection has entered through the lower extremities. Accidental inoculation of plague organisms into prisoners in Manila was made by the introduction of a hypodermic needle into the arm in the vicinity of the deltoid. In the majority of cases the first physical signs of the disease were the characteristic bubonic swellings of the inguinal region. From the primary bubo the infection may travel up the lymphatics and produce secondary buboes in other chains of glands. In other instances it is also quite possible that the bacilli may gain direct entrance to the blood vessels through injury to the walls of the veins in primary buboes.

In the pneumonic variety transmission probably takes place by direct infection through the respiratory or alimentary tract. Strong[3] and his co-workers, Teague and Crowell,[4] showed that the primary infection in pneumonic cases was apparently in the bronchi, and by extension into the lung tissues. According to Simpson,[5] infection may also take place through the intestinal tract by the consumption of plague-infected food. But at best this must be a very rare way for the disease to be transmitted. Blood cultures made during the febrile stage of the disease are almost always positive for the plague bacillus.

Morbid Anatomy.—The most characteristic feature in connection with plague autopsies is the bubo, of which over 50 per cent. are found, primarily in the femoral region. There is considerable suggillation in the dependent portions of the body, which has the appearance of large black spots. It is this sign which led to the name "black death." It is also quite common to find very small hemorrhages into the skin which are frequently not found on the autopsy table unless their presence has been noted in the living case. At times there are very small nodules about a millimeter in size which upon incision exude a turbid fluid which contains plague bacilli. The primary bubo is found in the lymph glands which drain the area of the skin which forms the portal of entry of the plague bacillus. The glandular mass feels boggy and elastic. The individual glands cannot be readily palpated. On section these buboes are found to be very edematous and exude a large amount of yellowish fluid. The appearance of a plague bubo is very characteristic. A large amount of the fluid exudes as soon as the skin over it is incised. Its edematous character distinguishes it readily from buboes due to other infections. Plague bacilli disappear from the glands usually about the time pus formation starts. The lesion in the gland is a hemorrhagic inflammation and coagulation necrosis. Other glands located along the lymph channels are also involved. In severe septicemic cases all of the glands may be enlarged and swollen, but large buboes are uncommon. Secondary plague lesions occur in the lungs, but these may be distinguished from primary pneumonic cases. The lesions in the lungs may be bronchopneumonic in character. There may be peripheral infarcts, and the metastatic type of infection. There may be punctiform hemorrhages of the pleura. The spleen is usually enlarged. The capsule is tense, opaque, varying in color from a reddish to a bluish appearance, and is often studded with small confluent hemorrhages. On section the spleen does not collapse or lose its form. Its consistence is firm. The cut surface is grayish-red in color and rather dull in appearance. There are usually small infarcts and necrotic nodules. Cultures from the spleen are usually positive. The liver shows changes found in acute parenchymatous inflammation. The changes in the kidney can scarcely be said to be characteristic, and are those usually found in acute febrile conditions. There is often degeneration of the tubular epithelium. The heart nearly always shows parenchymatous or fatty degeneration in the myocardium. The endocardium, as well as the myocardium, shows small hemorrhages at times. The most common lesion is a small hemorrhage in the epicardium.

Epidemiology.—A large majority of cases of plague occur singly in houses. When more than one case occurs in a house, the attacks are generally simultaneous. Plague may be conveyed from place to place by imported rat fleas which are carried by people on their persons or in their baggage. The human agent himself may escape infection. Often there are rats in containers of cargo, thus making it a simple matter to pass infected rats aboard outgoing steamers. Insanitary conditions have no relation to the occurrence of plague except in so far as they favor infestation by rats. A non-epidemic season is bridged over by acute plague in the rat, accompanied by a few cases in human beings.

Plague does not flourish with equal virulence throughout the different seasons of the year.[6] For instance, for more than 20 years the annual plague curve of Hongkong has reached its fastigium during May and its low point during December. In India the plague curve rises in July, reaches its high point in April, falls very rapidly until May, and is lowest in June. This seasonal variation has not been satisfactorily explained. It is sometimes ascribed to the variations in rat or flea breeding at different times of the year. Observations made in Java showed clearly that the average number of fleas per rat was very much higher during the greatest incidence of plague than during its lowest incidence. In India the number of cases seems to be associated with the humidity, the greatest number occurring during the years of the highest humidity.[7] Others have stated that when the temperature becomes lower than 50°F., rats with plague die before the plague bacillus reaches the peripheral blood, hence the fleas have no opportunity to become infected by biting, and are therefore incapable of conveying the disease. Satisfactory proof of this contention is lacking.

There is considerable difference in the plague transmission rate among the various species of rats, as well as in the rats of a given community. For instance, the Indian Plague Commission[8] found that of 1766 plague infected rats caught in Bombay, 1334 were Epimys Norvegicus, and that during the non-epizootic period the E. norvegicus was the only rat in which plague could be found, although this species is not nearly so numerous as the E. Rattus. The E. Norvegicus usually had about double the number of fleas found on the E. rattus. The E. norvegicus is more common in compounds, stables, warehouses, and grocery stores. The E. rattus is frequently found in sewers, drains, and stables, and seldom in houses above the third floor. It is especially noted for its burrowing proclivities. Creel[9] also found the E. norvegicus the most common in Porto Rico, although the infection rate of the E. rattus was half as high, as may be seen in the following table:

CLASSIFICATION OF ALL RODENTS EXAMINED IN PORTO RICA FROM JUNE 23 TO JANUARY 11*

		Per Cent. Infected
Norvegicus	23,453	0.0017
Rattus	4,201	0.0009
Alexandrinus	5,962	0.00016
Musculus	5,137	
Mongoose	233	
Unclassified*	309	
Total number examined	39,295	

* The unclassified were those examined during the first week of laboratory examination.

Among 68,667 rats caught in Manila during 1912 and 1913, the proportion of the different species was about the same as in Porto Rico.

The Indian Plague Commission showed that transmission from rat to rat is by means of the rat flea. Cages were prepared in which infected and healthy rats were only separated by a fine mesh gauze to prevent the transmission of fleas, and no infection took place. In other cages in which no provision was made to prevent the fleas from transferring themselves from sick rat to healthy rat, infection invariably took place. Healthy rats in cages hung 4 in. above plague infected rats did not contract the disease, although no wire screen was used. This was due to the fact that a flea does not jump higher than 4 in. When the cages were lowered to within jumping distance of the infected fleas, the healthy rats contracted plague. Many variations of this experiment have been carried on by the Indian Plague Commission and other observers, and the work has now been so thoroughly confirmed that practically no doubt exists.

The flea directly concerned in the transmission of plague is the one usually found on rats, namely, the Pulex cheopis, although other fleas are believed to be capable of conveying the disease. The exact method by which fleas transmit plague has not yet been definitely established. At first it was assumed that in the act of biting the human host, the plague flea injected plague organisms. Other observers were of the opinion that infection was conveyed through defecation of the flea at the time it bit its victim, and the itching caused by the flea-bite afforded an opportunity for the organisms to be rubbed into the skin. More recent work done at the Lister Institute shows that fleas usually regurgitate plague organisms contained in the sucked blood from the rat, and in this way the skin of the human host becomes infected.

The Indian Plague Commission believed the spread of plague to be due to fleas conveyed in merchandise or on human beings, rather than by the migration of rats. This conclusion is open to considerable doubt because plague fleas removed from their host are very short-lived, probably not more than a few days, and even a shorter time under unfavorable conditions such as sunlight. A human case probably does occur here and there in a community as the result of an infected flea having been introduced, but experience indicates that a sustained outbreak does not occur unless the rats become infected, and the weight of evidence is in favor of new localities becoming infected through the introduction of plague rats. Experience in Manila showed that practically every case of plague could be identified more or less directly with fleas that were not far removed from a plague rat. But this direct connection only became apparent after considerable experience in locating plague rats. A dead plague rat usually could be found in a ceiling with cracks, or in a floor with openings, or in a hollow wall close to a bed in which the human victim slept. It is assumed that the plague flea, not finding another rat for its host, seeks a human victim. In Manila, by placing healthy guinea pigs in the bed or room in which the human victim died, it was also possible to prove that fleas were responsible for the transmission of the disease. In the course of four or five hours, rat fleas would be found on the guinea pigs. If the fleas were permitted to remain, the guinea pigs often died of plague.

There is much reason to believe that so long as fleas can find rat hosts, they will not attack human beings.

In tropical countries bamboo structures have been found to be directly associated with the spread of the disease. This is due to the fact that the round bamboo joints make admirable harboring places for rats. As bamboo is used for beds and other articles which are used in close proximity to man, when rats die of plague the fleas have an opportunity to find a human victim. For instance, in some sections of

Manila and in many parts of Java, plague was eradicated by cementing the end of bamboos used in furniture, house, or fence construction.

Pneumonic plague probably spreads directly from man to man. In the outbreak in Manchuria, for instance, many doctors and nurses were victims of the disease, but as soon as proper masks were used by those caring for the sick, the infection among attendants ceased. Strong and Teague found plague bacilli in droplets of mucus expelled during coughing, or sometimes even during speaking. Pneumonic plague is more likely to spread in cold countries than in warm countries. Recent observations (1923) indicate that the inciting cause of primary pneumonic plague may not be the bacillus of bubonic plague or that symbiotic organisms may be necessary to start a plague pneumonia. It is however reasonably well established that the approach of warm weather is accompanied by a rapid decline in the number of cases of pneumonic plague.

There is little difference in sex incidence of plague, except in so far as persons are likely to be exposed to rats or to humans who are suffering with pneumonic plague. There is a general impression that the incidence of pneumonic plague is less among children. When the lungs are involved, especially in cases located in poor hygienic surroundings, its transmission directly by droplet infection from human to human is quite conceivable.

In California, a more or less permanent reservoir for plague exists in ground squirrels. Elaborate measures have been taken to free whole counties of squirrels, and on account of the extensive work done, transmission to human beings seldom takes place.

Outbreaks of human plague are almost invariably associated with considerable mortality among rats. In communities which have a modern health service it is customary to make periodical examinations rats in order to determine whether plague exists among them. If it is found, adequate steps are at once taken to exterminate rats and to bring the disease under control. In this way human outbreaks have been reduced to small proportions, and not infrequently have been entirely prevented.

Prophylaxis.—The control of plague depends upon preventing rat fleas, that have bitten plague infected rats or human beings, from biting humans. Bed bugs and other vermin may be largely disregarded. To accomplish this means killing rats or banishing them to places remote from human contact. The spread of human plague is probably greatly retarded by the fact that rat fleas do not bite man by choice, but only after failure to gain lodgment on rats. It thus happens that many persons contract the disease who sleep in the neighborhood of places infested with plague rats. In countries in which bamboo enters into the construction of houses, particularly beds and furniture, plague is often indirectly spread through this means. Rats often nest in bamboo joints, and when they die of plague the fleas seek other food sources if there are no more rats. If cats are convenient, they suffer. If not, humans are attacked. Very often persons who sleep in beds made of bamboo or in the vicinity of bamboo structures are the victims. Hollow walls in which rats die are also often directly concerned in transmitting plague to persons. Likewise persons who sleep on floors below which there are hollow ceilings, affording harborages for rats, are very frequently infected. Persons who go about barefooted and barelimbed in the vicinity of rat nests which have been inhabited by plague rats are very liable to infection. It is quite noteworthy that for years it was known that persons who worked in oil warehouses, or soiled their clothing with kerosene, seldom contracted the disease. The reason for this, of course, is now quite obvious. Fleas dislike the smell of kerosene, and die if they come in contact with it.

From the foregoing it will be apparent that the prophylaxis readily divides itself into two classes: Public and personal.

Public Prophylaxis and Control.—This consists in preventing and destroying rat breeding-places in the vicinity of man's habitations. This is usually brought about by eliminating hollow walls, hollow ceiling, posts, etc. Rats are naturally shy animals, and if their means of hiding are taken away they are inclined to leave human habitations. In most countries in which plague is liable to prevail, especially in port cities, ordinances are being gradually adopted which prevent the construction of buildings which are not reasonably rat-proof. This also applies to docks and piers, so that rats coming from ships that have touched at plague-infected ports may not find harborage on the piers. During the presence of an outbreak of plague, the primary measures for combatting the disease consist in destroying the rats and preventing their harborage near man. For instance, if the problem concerns rat control in a city, the method employed by the author in Manila has proved very successful:

A list was made of the places in which plague-infected rats were found. Each was regarded as a center of infection. Radiating lines, usually five in number, were prolonged from the center, evenly spaced like the spokes of a wheel. Rats were caught along these lines and examined Plague rats were seldom found more than a few blocks away. The furthermost points at which infected rats were found were then connected with a theoretical line, and the space enclosed was regarded as a center of infection. The entire rat catching forces which had heretofore been employed throughout the city were then concentrated along the border of the infected section. They then commenced to move toward the center, catching the rats as they closed in. Behind them thorough rat proofing was carried out. The use of this method, theoretically at least, prevents the spread of plague rats in a city. It affords the best chance to localize the infection. The plague infected rats are destroyed before they have an opportunity to spread the disease. One section after another was treated in this way until the rats had all been destroyed. Once weekly thereafter rats were caught in the previously infected sections and at other places which were insanitary and which had been infected in years gone by. This continued for one year.

Methods of Destroying Rats.—Rat trapping or poisoning is usually employed. The relative effectiveness of various rat traps shows that a wire spring or snap trap has an efficiency of 7.47 as against 0.97 for the wire cage trap, and 0.12 for poisoned bait. Many different forms of rat poison are advocated. The one found most successful in Manila consisted in boiling one part white arsenic with four parts (by weight) of rice, and distributing this about in places frequented by rats. One kernel of this poisoned rice is sufficient to kill a rat. Rats tire very quickly of a given bait and it should be frequently changed. A phosphorus rat paste often gives good results.

A plan for catching large numbers of rats in different parts of the city must be worked out to suit local conditions. A laboratory must be established for autopsying the rats. Careful examinations will soon show which sections of the city are infected, and rat-proof construction can be begun there.

In seaports precautions must be taken to prevent infected rats from gaining access to vessels, and also to prevent rats from other ports from gaining access to the shore. This may be accomplished in part by the use of rat funnels on the lines leading from the vessels to the shore, by fending the vessels 3 feet or more from the dock, and by lifting the gangways when not in actual use and the fumigation of cargo likely to harbor rats. A more thorough method is the fumigation of such ships at frequent intervals. This may be done with a 2 per cent. sulphur dioxide gas. Carbon monoxide and carbon dioxide have been recommended from time to time, but are not regarded as satisfactory because they only kill the rat and the flea escapes. An apparatus devised by Harker uses flue gases from steamers or launches. This proved very successful in killing rats, but failed to kill fleas. Probably the most successful agent is hydrocyanic gas, but it is extremely dangerous and many fatalities have already been recorded from its use. Recently a tear gas has been added which it is hoped will give warning to persons who are coming into dangerous proximity of hydrocyanic gas.

Taking everything into consideration, the safest method for killing rats on board ships, under average conditions, is with sulphur dioxide. This can be readily generated in iron pots (Dutch ovens) that have been set into tubs of water. The sulphur is ignited, the holds, cabins, and rooms are tightly closed, and the gas is allowed to remain for a period of at least six hours. Two pounds of roll sulphur for each 1000 cu. ft. of space are sufficient. Rat guards should be used on the lines by which vessels are tied to wharves, in order to prevent rats from passing back and forth. At night the gangway should be lifted. Vessels from plague-infected ports should also be fended away from the wharf for a distance of 3 feet at least.

The rat virus which has been recommended from time to time for the purpose of spreading fatal disease among rats has proved very disappointing. It soon loses its efficiency, and when it does act it often obscures the diagnosis of plague among rats.

In connection with the foregoing measures, provision must be made for isolating human cases, and adequate precautions taken to prevent fleas with which they may have been infested from gaining access to healthy individuals. This can be done by placing all the patient's clothing and bedding in bags, and disinfecting them in a steam chamber in vacuum. A temperature of 110°C. should be maintained for at least 20 minutes. Attendants who handle such infected clothing should be dressed in flea proof suits in which the neckbands, wrists and ankles are snugly fitted with elastic or other means to prevent the entrance of fleas. It is also deemed advisable to disinfect the sputa or excreta which come from the plague sick. A bacteriological laboratory where diagnosis may be reliably and satisfactorily made is an absolute essential in dealing intelligently with plague outbreaks.

Personal Prophylaxis.—This consists in avoiding infected regions and guarding against flea-bites. For those who are compelled to enter plague-infected areas, in addition to wearing flea proof clothing, some measure of precaution may be obtained by sprinkling kerosene or some other insecticide about the tops of the shoes, wrists, and neckbands. Haffkine's and other vaccines are said to give protection for a period of at least six months and perhaps longer, and those who are constantly exposed to plague fleas should protect themselves in this way. So-called true vaccines, that is, those made with attenuated living plague bacilli, are generally held to be more effective than those made with dead bacilli, as is done with the Haffkine method. Those who come in contact with pneumonic plague should wear face masks which may be made of gauze with suitable eyes of celluloid. Extensive experiments made in the United States during the influenza epidemic of 1918 showed that in order to prevent the passage of ordinary respiratory bacteria, at least 325 strands to the square inch of ordinary surgical gauze should be placed before the nose and mouth. This may be accomplished by using eight layers of 20 by 24 gauze $(20 + 24 \times 8 = 352)$ or three layers of 40 by 44 gauze (cheese cloth) $(40 + 44 \times 3 = 336)$. Cheese cloth or more closely woven material is said to give better results than numerous layers of loosely woven material.

For several years after plague has disappeared from a city it is well that examinations of a limited number of rats should be made at weekly intervals, particularly rats from areas in which plague infection has prevailed. Effective work in this direction and adequate measures taken when plague infected rats are found will probably prevent outbreaks among human beings.

Clinical Course, Symptoms, and Duration of Disease.—The incubation period of plague varies from 2 to 10 days. In most cases it occurs within a period of three days. For clinical purposes the disease may be divided into four types: Pestis minor, bubonic, septicemic, and pneumonic.

Pestis Minor.—In this type of plague the patient has an irregular fever, swelling in the glands of the groin, and possibly suppuration. Very often the patient is not ill enough to seek relief. These cases are commonest at the beginning and end of epidemics. Plague bacilli can usually be found in the glands and in the blood if sufficiently large quantities are taken during the height of the fever. Diagnosis can often be arrived at by searching for the typical plague vesicle which probably denotes the entrance of the infection, and finding therein the plague bacilli. The impression prevails that this type is more frequent among children than among adults.

Bubonic.—There may be prodromal symptoms, consisting of general malaise, headache, pain in the back, uneasiness, chills, and mental apathy. This variety occurs in probably three-fourths of all the cases. The onset is sudden, often with chilly sensations, but there is seldom a distinct rigor. The temperature rises to 103 (39.5°C.) or 104 (40°C.), with a corresponding increase in the pulse and respiration rate. There is usually a marked increase in the prodromal symptoms. The mental dulness is very characteristic and often leads to suspecting the disease. The patient's expression is that of fear and anxiety. The eyes are bloodshot, bright, and staring. The face is drawn, and often the nostrils are dilated. The temperature may remit somewhat on the second day, but usually rises again almost immediately afterward. If recovery takes place, the fall in temperature is usually by lysis. In fatal cases the temperature often falls rapidly to normal or subnormal and then rises quickly to 107 (41.5°C.), followed by death. Glandular swellings become prominent on the second day and very often can be felt by careful palpation on the first day. If the patient survives until the fourth day, the swellings are usually quite large and distinct. Suppuration may occur, or in some instances, gangrene. Suppuration is usually not regarded as a favorable symptom. Petechiæ usually appear about the third day. These are often referred to as "black spots" or "tokens of death," and gave to the disease the name "black death" in the middle ages. Hemoptysis frequently occurs. Plague bacilli can usually be found in the blood during the high fever periods of the disease. There is a leukocytosis from 90,000 to 100,000, and the red-cells and the hemoglobin are distinctly increased. The increase in the white count is due almost entirely to the polymorphonuclear leukocytes. The breathing is rapid, the breath sounds harsh, and there are generally moist râles.

The urine is usually diminished in amount, and contains albumin and casts. Pregnant women generally abort. There is low muttering delirium which gradually passes into coma. Death usually occurs between the third and fifth days. In favorable cases the tongue becomes moist, the pulse rate and temperature fall, and delirium gradually abates. Even in favorable cases buboes generally continue to enlarge and soften. If not incised, they usually burst spontaneously. As a rule they are very ill-smelling. In these cases convalescence usually begins between the sixth and tenth days.

Septicemic.—In this form of the disease the patient usually succumbs within three or four days and before the appearance of buboes. The symptoms are similar to those of any general septicemic process. The degree of virulence and rapid course of the disease depend on the entry of large numbers of bacilli into the blood. The patient is extremely prostrated from the beginning. He is pale and apathetic, and as a rule there is no great febrile reaction. The temperature may not be above 100°F. Vomiting is severe, and diarrhea with blood frequently occurs. There are often petechial hemorrhages in the skin. Leukopenia is the rule in these cases. Briefly, there is an overwhelming infection in which the patient succumbs before the defensive forces of the human organism have had an opportunity to act.

Pneumonic.—True pneumonic plague is a primary infection and must be carefully distinguished from the ordinary inflammation of the lungs found secondary to bubonic plague. The disease begins suddenly with fever, shortness of breath, coughing, and frequently pain in the chest. There is expectoration of bloody mucus which contains plague bacilli. Cyanosis comes on early. The pulse is small and rapid. There is early enlargement of the spleen, and death usually occurs within three days. Sixty per cent. of cases die within 48 hours, 16 per cent. within 24 hours, and 16 per cent. within 72 hours.[10] In some instances persons with pneumonic plague topple over dead while at work or while undergoing some sudden physical strain. The diagnosis of this form of the disease in Chinese has been confirmed a number of times by autopsy. The incidence among children appears to be less than among adults. Recovery in this type of the disease is very rare. The other symptoms correspond very closely with those of pneumonia caused by the pneumococcus or other organisms.

Complications.—Complications in plague are extremely rare, and even in the few cases that recover there are very few sequelæ. Gangrene of the lung is occasionally present. Spontaneous rupture of buboes occurs from time to time, but this is really a process directly concerned with the disease.

Prognosis.—The mortality from plague is probably higher than from any other epidemic disease that occurs in considerable numbers. As a rule the mortality varies from 80 to 90 per cent., and many outbreaks have had a mortality of 95 to 98 per cent. The prognosis depends very largely on the character of the outbreak, that is, the virulence of the strain of plague bacilli. In the pneumonic form of the disease the recorded mortality in reliably diagnosed cases is 100 per cent. The septicemic type has a higher mortality than the bubonic variety. A study of the age incidence and mortality shows that they vary but little in the different age groups.

Diagnosis.—The first cases in a community in which plague is rare, or has not heretofore appeared, are very likely to be overlooked. But once the presence of the disease is suspected, its diagnosis is comparatively simple and can be made practically certain by resort to laboratory methods. Very often the disease is mistaken for typhoid fever, but the appearance of the buboes, and finally the demonstration of the plague organisms, makes the distinction certain. The pneumonic type is very likely to be overlooked and very often only attracts attention through nurses and doctors contracting the disease. In the tropics plague is often confused with ordinary glandular fever, and during epidemics many cases are sent to plague hospitals by mistake. The clinical examination alone will usually suffice to distinguish the disease with reasonable certainty. In glandular fever the glands are exceptionally hard and movable, whereas the plague bubo is more in the nature of an edematous mass in which it is difficult to palpate the individual glands. There is very little prostration in glandular fever, whereas in plague there is very great prostration as a rule. Bacteriological diagnosis based upon material taken from the gland or by direct examination of the blood may be depended upon to show plague. In conjunction with the clinical symptoms, the presence of the typical bipolar staining bacilli is almost sufficient evidence upon which to make conclusive diagnosis. An absolute diagnosis must be dependent upon blood cultures and animal experiments. Diagnoses may be confirmed by inoculating rats or guinea pigs with blood or material taken from a bubo, and upon the test animal becoming sick, further inoculations from that animal into a healthy animal, which should cause the disease. In the majority of cases of pneumonic plague, death occurs in 48 hours, which is in sharp contrast to influenza and other pneumonias.

Treatment.—There is no specific for plague. Sera have been made in various ways from time to time, but none of these can be said to have any appreciable influence on the mortality of the disease, unless they have been given during the prodromal or incubation period. The treatment resolves itself largely into making the patient comfortable and following the same general principles which apply to the care of fever patients. There should be purgation and stimulation, with the use of morphine to control the pain. It is customary to apply ice compresses to the buboes. With regard to the use of serum, while there is no satisfactory evidence that it is of value, and there is considerable evidence that it does no harm. Fever may be controlled by sponging and cold applications. If the buboes break down, they should be incised and have antiseptic treatment. Ichthyol is frequently recommended for this purpose. The heart should be sustained by digitalis and strychnine. There should be administration of sufficient fluid to keep the kidneys active. The diet should be of the liquid variety. Milk is usually well borne. The different mortality results are probably not so much due to the effect of treatment as to difference in dose, virulence of the plague organism, and the resistance of the individual.

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CHAPTER CXXVII

ACTINOMYCOSIS

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Definition.—Actinomycosis is a subacute or chronic suppurative process occurring chiefly in cattle, but also affecting man, caused by a specific microörganism, Actinomyces bovis, or "ray fungus."

History.—The knowledge of actinomycosis as a specific disease dates from the researches of Bollinger in Germany, who, in 1877, established the ray fungus as the cause of the disease. To Harz we owe the name "actinomyces bovis," so named because of the ray-like structure of colonies of the microörganism. J. Israel described the disease in man, but did not recognize the causal fungus as identical with that described by Harz in cattle. Ponfik,[1] in 1879, first pointed out the identity of bovine and human actinomycosis. Murphy of Chicago, in 1885, reported the first cases of human actinomycosis in the United States.

Distribution.—The disease has a wide geographical range and frequently has been reported both in man and cattle from Germany, Austria, Switzerland, England, Russia, France and the United States; less frequently from Denmark, Roumania, South America and elsewhere. Apparently the disease is more common in Germany than in England and the United States.

Incidence.—Actinomycosis is observed chiefly during adult life although it is of occasional occurrence in childhood. On the whole it must be said that actinomycosis is relatively rare during infancy and childhood. In Erving's[12] analysis of 100 American cases the youngest was a child of six years. No other case was reported under 10 years of age. The disease occurred 9 times in individuals ranging from 10 to 20 years.

It is most common in the middle decade of life, is said to occur oftener in the cities and more frequently in the months from August to January.

Etiology.—The specific fungus occurs in the lesions and pus as small granular yellowish or grayish masses averaging 1 to 2 mm., which when examined microscopically are seen to consist of interlacing filaments and rods and coccoid bodies. with peculiar hyaline swollen bodies arranged around the periphery in a more or less radiate fashion. These highly refractile bodies are spoken of as "clubs" or "rays" and are best seen in granules from bovine lesions. In human lesions, "clubs" frequently are entirely absent and the colony consists solely of a central mass of irregularly interlacing filaments, degeneration bodies, rods and coccoids and radiately arranged filaments around the periphery. The center of the granule may consist entirely of broken-down filaments and necrotic material. As a general rule, the granules from human lesions are softer in consistency than those from lesions in cattle. In the latter, the granules not infrequently show calcification.

The cultural characters of Actinomyces bovis have afforded apparently unending controversy and confusion. More broadly, this statement holds true for the classification and relationship of pathogenic fungi in general.[2, 3]

The microörganism first described by Wolff and Israel[4] and later by Wright,[5] Harbitz and Grondahl,[6] Henry,[7] Stokes[8] and others, probably is the exciting cause of actinomycosis in man. It does not grow readily upon ordinary media, develops best anaërobically at body temperature, exhibiting little or no growth at room temperature. It does not produce spore-like bodies, as described by Bostroem,[9] but certain strains, especially those from bovine lesions, produce the "characteristic" clubs in cultures. According to Wright,[5] the best growth is obtained in deep dextrose (1 per cent.) agar tubes.

Experimental intraperitoneal inoculation of pure cultures of Actinomyces bovis into guinea pigs and rabbits has yielded rather inconstant results. The typical nodular lesions of actinomycosis have been produced in healthy experimental animals, but in no case has an active, progressive infectious process been set up. The results of 35 investigators, reviewed by Wright,[5] show absolutely negative results in about one-half of the experimental inoculations.

The microörganism first described by Bostroem[9] as the cause of actinomycosis is considered by Wright and other workers either as a secondary invader or extraneous and accidently introduced into the cultures. It is described as growing rapidly and luxuriantly at room temperature on all ordinary media. It is an aërobe and produces, according to Bostroem, spore-like reproductive elements out of the substance of the filaments. Animal inoculations with this group have been entirely negative.

Modes of Infection.—Wright believes the microörganism has its normal habitat within the body. He says: "The specific microörganism of actinomycosis probably exists normally among the abundant flora of the secretions of the alimentary tract, where it has not been recognized because it exists there in a form which is not characteristic and very unlike that which it assumes in the lesions; it may gain entrance to the tissues through wounds, made by penetrating foreign bodies or otherwise, or through lesions due to carious teeth, or may invade the lungs as do other bacteria of the mouth and pharynx; in the tissues under certain conditions it develops into the characteristic colonies or granules and by further proliferation gives rise to the disease. The disease in and about the jaw is frequently associated with carious teeth[10]. Infection of carious teeth and tonsillar crypts with actinomyces has been reported by Lord.[11]

The frequently accepted idea that the natural habitat of the microörganism is on grains and grasses and that infection occurs through penetration or entrance of such infected foreign bodies into the tissues seems to be ill-founded, because the true Actinomyces bovis does not possess saprophytic characters. The saprophytic fungi isolated from grains and grasses more closely correspond with the microörganisms Bostroem claimed to have isolated from actinomycotic lesions in cattle.

Pathology.—"The essential effects produced in the tissues by actinomyces are suppuration and tissue destruction, combined, in most cases, with new formation of granulation and connective tissue. In some cases the new formation of connective tissue may be excessive in amount and thus simulate a neoplasm. The granulation tissue usually contains many large cells filled with fat globules. Giant cells may also be present" (Wright,[10] p. 331).

The pathologic process extends both by continuity of tissues and by metastases, usually by the blood stream. Extension by continuity results in greater or lesser portions of a tissue or organ being converted into pus cavities surrounded by connective and granulation tissue. In these abscesses, actinomyces granules and sometimes pyogenic bacteria are found. These suppurative tracts frequently possess sinuses which open either on the skin or in mucous membranes. Healing may occur by cicatrization and complete death of the enclosed microörganisms. Metastatic extension occurs when a local actinomycotic process ruptures and the microörganisms are discharged into the blood stream. Wide dissemination may occur, the lungs or liver usually being primarily affected and secondarily the heart, brain and other organs.

Tumor-like actinomycotic formations more frequently are met with in cattle than in man, but in man sinuses and fistulas are more common.

Clinical Types.—Actinomycosis of Head and Neck.—Primary actinomycosis in this region is the most common, more than 50 per cent. of all the recorded cases in man being of this form (Wright[10]). The oral and pharyngeal cavities offer by far the most common portal of entrance for the fungus, which may affect the tongue, the larynx, the skin of the face and neck, the bones of the face and skull, and the brain.

Abdominal Actinomycosis.—In man, this group constitutes about 20 per cent. of the cases. Abdominal actinomycosis may arise primarily by infection from the intestines, or by secondary extension or metastasis from elsewhere in the body. The cecum and appendix offer the most usual primary focus, from which the extension of the disease between the coils of the intestines to the abdominal wall is marked by fibrous adhesions and abscesses. The muscle of the abdominal wall suffers connective-tissue replacement, within which are found pockets of pus, sinuses and intestinal fistulæ.

Any abdominal organ or region may be affected. Single or multiple suppurative foci in the liver are frequent. Involvement of the spleen and kidneys and direct extension of the disease process to the thorax or pelvis and extremities may occur.

Thoracic Actinomycosis.—This group comprises about 15 per cent. of the cases, the most frequent site of the disease being the lungs. Here abscess and cavity formation occur, with more or less extensive replacement of lung tissue by fibrous connective tissue. Involvement of the inferior lobes of the lungs is more frequent than that of the superior lobes. Extension of the disease process into the mediastinum may occur, either from cervical lesions above or from abdominal lesions below, and involvement of the lungs and thoracic wall occur secondarily by extension.

Cutaneous Actinomycosis.—Primary actinomycosis of the skin is very rare. It is an ulcerative process, most often resembling lupus or syphilis, and has a very chronic course. Most frequently, cutaneous actinomycosis is secondary to infection elsewhere in the body.

Two cases occurring in childhood are cited by F. Karewski.[13] A boy of five who had swallowed a kernel of barley developed pain, dysphagia, and hematemesis. A swelling developed between the vertebral column and the right angle of the scapula. This was opened and discharged a portion of the barley kernel which had been swallowed. New abscesses developed which contained ray fungi and a diagnosis of actinomycosis was made. In spite of energetic operative treatment the abscesses continued to spread. Pulmonary symptoms ensued. The child's general condition suffered, carious destruction of the vertebræ developed, and the child died.

In the case of Bostroem a tumor of the right clavicular region developed in a girl of eleven. It spread to the right thoracic wall and healed after incision. A year later an abscess developed in the iliac fossa without involvement of the bone. It healed after nine months. Later the child developed a pericarditis with collection of pus below the heart. Multiple metastases developed and the child died. The pus showed actinomyces. The fungi probably passed through the gastro-intestinal canal into the mediastinum and loose tissue of the neck, extending to the clavicle and ilium, as well as to the pericardium and other organs by the prevertebral route.

Diagnosis.—The only definite diagnostic test is the finding of the actinomyces in the pus or tissues. This may be accomplished in several ways, (1) microscopic examination of the pus in the fresh state; (2) examination of smears stained by Gram's method or tissues stained by Gram's or Mallory's method; (3) cultivation of the microörganism upon bacteriologic media.

Prognosis.—In man, spontaneous recovery is rare. The prognosis largely depends upon the localization and extent of the disease. Actinomycosis of parts surgically accessible, such as the skin, jaw, etc., and more especially actinomycosis of the head, offers the best prognosis. Erving[12] reported that out of a total of 53 American cases of actinomycosis of the head analyzed by him, 36 recovered and 5 died. Prognosis in the thoracic and abdominal forms is bad. Of 20 American cases of thoracic actinomycosis, 15 died and 2 recovered and of 23 American cases involving the abdomen, 10 died and 5 recovered (Erving).

Treatment.—The best treatment, if possible, is surgical, combined with the internal administration of potassium iodide, one to two grains daily, gradually increased up to four and five grains and continued over a long period of time. The drug probably has no specific action on the ray fungus as the organism has been found in the body tissues after months of systematic potassium iodide treatment. Treatment with *x*-rays, combined with the use of potassium iodide, has been recommended in cases not amenable to a radical surgical removal of the diseased tissues.

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CHAPTER CXXVIII

GLANDULAR FEVER

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Definition.—Glandular fever is an acute infection of children, of undetermined etiology, characterized by an inflammatory swelling of the cervical and other lymphatic glands, associated with fever and constipation, and followed by a considerable degree of anemia and general prostration.

Etiology.—**Seasonal Incidence.**—In the epidemic described by West,[1] the largest number of cases occurred in October. No cases occurred in June, July, or August, and but one case in September.

Age.—The disease is one of children, is not uncommon in infancy, but is very rare after puberty. West's[1] cases ranged in age from 7 months to 13 years, 56 of the 96 children affected being five years of age or younger. Donkin[2] has described several instances in which young adults were attacked during the course of a family epidemic.

Exciting Cause.—The exciting cause of glandular fever is unknown. Since the original article of Pfeiffer[3] (1889) practically nothing has been added to the description of the disease nor has any definite light been thrown upon its etiology. Two theories are held, first that it is an auto-intoxication, possibly of intestinal origin, and second that it is a specific infection. Comby states that it may be a streptococcus infection, its entrance being effected through the tonsils without any local lesion. In a case studied by him, Rudolph[4] isolated in pure culture a streptococcus from a suppurating cervical gland. The importance of the streptococcus as the exciting cause of the disease is open to suspicion, especially in Rudolph's case, in view of the fact that in glandular fever, the glands show little tendency to suppurate; "indeed, it is very doubtful whether this accident ever happens" (Williams[5]).

Pathology.—Pfeiffer,[3] in his original description of the disease, noted the fact that it occurs in narrowly localized epidemics, but that in families in which one member is affected, most of the other children of the susceptible age in the family contract the disease. The history of the cases described by West[1] was of an epidemic character, that did not extend beyond a single house or family. West especially called attention to the absence of any regular spread of the disease.

One of the striking features of glandular fever is the fact that the throat usually is normal. Slight redness of the pharynx may occur, "but examination of the throat reveals no condition sufficient to account for the stiffness of the neck and dysphagia, nor for the pyrexia" (Williams[5]). In only four of the cases studied by West[1] was a severe pharyngitis present. Williams[5] says: "The place of the disease in nosology would appear to be near bubonic plague, to the milder forms of which (pestis minor) it bears some resemblance."

Symptoms.—The disease is one of sudden onset. Often with no prodromata, sometimes after slight malaise (Koplik[6]) children are attacked with fever, restlessness, headache, and pain in the neck, which is held stiffly, pain on deglutition and sometimes pain in the abdomen. The temperature, during the first two days, ranges from 101° to 103°F. The maximum fever (104°F.) usually is attained about the third day, after which the fever may fall by crisis (Koplik[6]) or may remain high as long as groups of glands enlarge (Williams). The pharynx, in the majority of cases, is normal. A slight cough may be present. In the majority of the cases, constipation is the rule, but in West's cases, diarrhea was present in almost all of the mild and shorter ones.

The most characteristic feature of the disease is the swelling of the cervical lymph glands. West claims that after a few hours of premonitory symptoms, swelling of the carotid glands begins. As a rule, the glands on the left side are involved first and the enlargement attains its maximum on the second to the fourth day. The swelling always is tender on pressure and sometimes painful. In no case, in West's epidemic, did the swelling of the cervical lymph glands appear simultaneously on both sides of the neck. In fact, the unilateral involvement of the cervical lymph-nodes, usually on the left side, at onset, seems to be characteristic of glandular fever. The swelling rarely continues unilateral. After the glandular enlargement on one side has reached its maximum, the glands on the opposite side of the neck usually become affected. The glandular swelling is uniform, as thick as an index finger (West) and is composed of several lymph-nodes. The glands may attain the size of a pigeon's egg. In three-fourths of the cases described by West, there was noticed enlargement of the post-cervical, axillary and inguinal glands, but never were they all enlarged in any one case, nor so much enlarged nor so tender as the cervical glands. In 37 cases the mesenteric glands could be felt enlarged (West[1]). In three quarters of his cases, abdominal pain and tenderness, usually in or below the umbilical region, was present. Out of 96 cases, in 87 the liver was enlarged and in 57 enlargement of the spleen occurred.

Course.—The incubation period probably is about seven days, with extremes of five and ten days (Williams). In West's epidemic, the duration of the disease varied from 9 to 27 days, and averaged 16 days. The duration of the most active symptoms usually is from four to seven days.

Diagnosis.—From mumps, the disease can be rather easily differentiated. "The swelling of the other glands, and of the liver and spleen, a crisis, and the continual swelling of the glands until the advent of the crisis, makes a picture very unlike that of mumps" (West[1]). In glandular fever, the parotid gland never is involved. From the glandular swellings occurring with grippal affections or pneumonia, glandular fever is differentiated by the fact that the lymph-nodes swell first on one side of the neck and then on the other.

"The opinion is expressed that many cases called febricula and influenza will, on close observation, prove to be glandular fever and that it occurs in a light form and, as such, is not often seen by physicians" (West[1]).

Complications and Sequelæ.—The convalescence sometimes is prolonged. The child becomes anemic and prostrated. Several months may elapse before complete recovery is established.

Complications are rare, the most frequent being nephritis, which may be attended by hematuria. "It is possible that many of the cases of acute nephritis which occur in children, apart from those due to exposure or scarlet fever, are sequels to unrecognized attacks of glandular fever" (Stark[7]).

Prognosis.—The prognosis is favorable. Out of the 96 cases described by West, there was death in but one, and that occurred in a delicate child convalescing from scarlet fever.

Treatment.—As the affection is self-limited and tends to spontaneous recovery, treatment is purely symptomatic.

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CHAPTER CXXIX

DENGUE

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Synonyms.—Breakbone fever, dandy fever, broken wing fever, giraffe or stiff-necked fever, etc.

Definition.—Dengue fever is an acute infectious disease of tropical and sub-tropical climates, characterized by two paroxysmal attacks of fever, intense headache, extreme joint and muscle pains, frequently skin eruptions. It is spread through the agency of a mosquito, *Culex fatigans*. It may occur at any age and has even been observed in the newborn infant.

Geographical Distribution.—The disease is of wide distribution. According to Coleman,[1] the name dengue is of Spanish origin, the disease being first observed in Spain during 1764–1768. Since 1780, pandemics have occurred in the United States, India and Spain, the epidemic in Philadelphia in 1780 being most graphically and completely described by Benjamin Rush. An epidemic in the United States occurred in 1897–1898 throughout Georgia, Florida and Texas. During the summer and fall of 1922, a widespread epidemic of dengue fever originated in Galveston, Texas, affecting 30,000 persons.[2] Subsequently, the disease spread throughout eastern and central Texas and into neighboring states. The number of cases in the State of Texas during this epidemic has been estimated by the State Board of Health at between 500,000 and 600,000.[3] Widespread epidemics have occurred in Java, India, China, Burma, Arabia, Egypt, Spain, Turkey, Greece, Syria, Isthmian Canal Zone, Peru, the distribution of the disease paralleling the distribution of the mosquito, *Culex fatigans*.

Etiology.—Under predisposing causes, the most important is climate. In the tropics there is no seasonal incidence of note. It is strictly a warm weather disease. "The ordinary limit of diffusion is generally placed at from 32° 47′ N. to 23° 23′ S. While this is the rule, it has occurred outside of this zone; e.g., in Philadelphia, New York, Boston and other places" (Coleman[1]).

The exciting cause of dengue is unknown. The specific, living microörganism apparently exists in the circulating blood, as intravenous injection of 20 c.c. of the blood of a patient suffering with dengue fever, sets up the disease in a healthy individual, after an incubation period of two or three days.

Cleland, Bradley and MacDonald[4,5] showed that the causative organism was present in serum and citrated blood; the virus remaining infective in blood outside the body at room temperature for four days, and in an ice-box up to seven days. Following the discovery by Noguchi of Leptospira icteroides in yellow fever, the opinion has been advanced that dengue would be found to be caused by a Leptospira. Chandler and Rice[3] report, in their study of the Texas epidemic of 1922, that dark field examinations of about 250 slides of fresh blood from about 70 patients, inoculations into guinea-pigs, mice and monkeys, and attempts to cultivate the organism in media employed by Noguchi, have all given negative results for Leptospiræ.

Graham[6] (1903) described as the etiologic factor in dengue an intracorpuscular "organism with amoëboid movement, and in many ways resembling the Plasmodium malariæ." Though his observations on the etiology of the disease have failed of corroboration at the hands of subsequent investigators, notably Ashburn and Craig,[7] in the same article Graham first definitely settled the chief, if not the only, mode namely, by the bite of mosquitoes. He was the first to experimentally infect human individuals by causing them to be bitten by the mosquito Culex fatigans, which previously had fed on dengue-fever patients, and also reproduced the disease in a human subject by inoculation of a solution made from the glands of infected mosquitoes. As regards the mosquito mode of transmission Graham's work has been fully confirmed by Ashburn and Craig.[7]

More recent experimental work has suggested that the Aëdes egypti is the principal, if not the only insect transmitter of dengue. Cleland, Bradley and MacDonald[4,5] successfully transmitted the disease by means of Aëdes egypti to 4 out of 7 human volunteers, but failed in 3 cases in which Culex quinquefasciatus (fatigans) was employed. Chandler and Rice[3] note the fact that the 1922 Texas epidemic was preceded, and accompanied by, an unprecedented scourge of yellow fever mosquitoes (Aëdes egypti). Transmission experiments carried out with Aëdes egypti were successful in 4 out of 6 human volunteers, the mosquitoes transmitting the disease in from twenty-four to ninety-six hours after feeding on patients in the second to the fifth days of the disease.

Pathology and Morbid Anatomy.—These are unknown. A considerable number of autopsies have been performed and no definite anatomical lesions have been found.

Symptoms.—After an incubation period, variously estimated at from two to six days, the disease is ushered in by a sudden onset, headache, intense muscle and joint pains, especially in the back and legs, and rapidly mounting temperature. The temperature varies from 100 to 107° F., cases of moderate severity averaging 103° to 104° F.

After the initial attack, which has an average duration of three days, the fever remits. Then within two to four days, there is a second attack of fever, which ordinarily lasts from a few hours to 24 hours and is accompanied by a return of the intense pain. In the majority of cases a rash appears, usually on the fifth or sixth day, showing first on the palms and backs of the hands, rapidly spreading up the arms to the trunk, thighs and legs. Desquamation follows the eruption The rash, most freqeuntly either measly or scarlatiniform, may persist for a number of weeks after recovery from the disease. The average duration of a moderate attack is from seven to eight days. Complications, when fatal, most frequently are peritonitis and pleuritis. Muscle atrophy and meningitis may occur. Muscle and joint pains are the most common sequelæ.

Diagnosis.—The disease may be diagnosed by its pandemicity and rapidity of spread, by the rather characteristic temperature curve the severe pain and sudden onset. According to Ashburn and Craig, dengue is characterized by a well-marked leukopenia, the polymorphonuclear leukocytes being decreased, with a marked increase in the small lymphocytes.

Prognosis.—Death is rare and occurs chiefly in small children who develop severe hematemesis and convulsions before death. Castellani and Chalmers[8] refer to one death in 1000 cases in Australia.

Treatment.—Treatment is purely symptomatic. Hydrotherapy is of value in controlling the fever, and morphia or Dover's powders in alleviating the pain which would otherwise be often intractable.

Prophylaxis.—Warfare, as in malaria, against the mosquito, most especially Culex (fatigans) and Aëdes egypti, sums up the question of prophylaxis.

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CHAPTER CXXX

THE TRYPANOSOMIASES

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AFRICAN TRYPANOSOMIASIS (SLEEPING SICKNESS)

Definition.—The African form of human trypanosomiasis, caused by Trypanosoma gambiense and Tr. rhodesiense, and conveyed by a fly, Glossina palpalis, is an extremely fatal chronic disease, characterized by glandular enlargement, irregular fever and, finally, by apathy, weakness, coma and death.

History.—Sleeping sickness has been known on the African West Coast for over a century (Bruce[1]). The earliest mention of the disease is said to be found in a book written by John Atkins in 1734, in which he refers to "the sleeping distemper" among negroes on the Guinea Coast (Castellani[2]).

The first description of the Tr. gambiense was given by Dutton,[3] who discovered the parasite in the blood of a British government official from West Africa, who had been invalided to England. In 1902, Castellani found the trypanosome in the blood and spinal fluid of natives in Uganda suffering with sleeping sickness and in 1903 he reported the trypanosome to be the etiological basis of the disease, based upon the fact that he found the characteristic trypanosome in the centrifugalized cerebrospinal fluid of 20 out of 34 cases of sleeping sickness.[4]

In 1910, Stephens and Fantham discovered a trypanosome, very similar to Tr. brucei, in the blood of sleeping sickness patients in Rhodesia and to this species they gave the specific name Tr. rhodesiense.

Etiology.—Geographical Distribution.—Originally noted on the Guinea Coast, sleeping sickness has followed along definite channels of inter-communication opened up by man until now the Tr. gambiense, first thought to be a harmless parasite in the blood of natives in Uganda, has taken its place in the annals of tropical medicine as the exciting cause of one of the deadliest scourges of man. Limited in its range, even as late as 1882, to Senegal, to Loando and the islands of the Gulf of Guinea, the geographical distribution of sleeping sickness rapidly widened as the white man pushed back further and further the barriers of "unknown" Africa. The travels of the explorer Stanley unquestionably were instrumental in introducing the disease into hitherto unaffected regions. The relief by Stanley and his Congolese of Enim Pasha and his people at Wadelai on the Victoria Nile and the subsequent deportation of Pasha and his 10,000 natives from the region of Albert Nyanza to Busoga and Uganda introduced into these two latter countries sleeping sickness, a plague which caused in Uganda alone the death of 100,000 negroes within three years of its introduction and is estimated in the Congo Region to have taken a toll of half a million natives between the years 1896 and 1906. By the year 1904, the disease was found by Dutton and Todd to have become widely disseminated throughout the Congo Free State. As trades routes have developed, the disease has passed from the Congo into eastern Rhodesia, northwards to Wadelai, on the Victoria Nile, and into the Sudan. Castellani[2] defines the distribution of the disease as extending "along the West Coast of Africa, from St. Louis in Senegal, to Mossamedes, in Angola; from the coast to Timbucktu on the Niger; through the whole of the Congo into Uganda and Rhodesia; from Uganda and Busoga southwards to German East Africa, and northwards into Bahr-el-Ghazal Province of the Sudan."

Race.—Race is generally regarded as having very little influence. It was formerly thought that the disease was confined to negroes, but numerous white men have succumbed to sleeping sickness. A sex or age predisposition has not been determined. Children may develop trypanosomiasis if exposed. Feldman treated a child from Uganda for sleeping sickness. Koch's report of the German expedition for the study of sleeping sickness in East Africa, 1906–1907, contained several photographs of children. Marked edematous swelling of the eyelids may be noticed in these photographs.

Occupation.—The influence of occupation is well described by Bruce;[1] "Whatever occupation leads a native to spend much time on the wooded shores of Victoria Nyanza or any river or lake within the sleeping sickness area, is dangerous. Hence fishermen, canoemen and the inhabitants generally of the lake shore are prone to suffer. These people are half-naked and spend much of their time trading and gossiping on the shore, 30 to 80 per cent. of them harboring the trypanosomes in their blood, and being constantly bitten by numerous tsetse-flies." Porters and other travelers passing through the fly-belts are very much exposed to infection.

Description of Causal Agents.—A plurality of species of trypanosomes causing sleeping sickness in man seems to be established. Tr. gambiense, Dutton and Todd, 1902, and T. rhodesiense, Stephens and Fantham, 1910, are the two species accepted.

Tr. gambiense varies rather markedly in size. It measures from 17 to 28μ in length and 1.4 to 2μ in breadth (Laveran and Mesnil). It may be as short as 14μ and as long as 33μ. In common with most trypanosomes, Tr. gambiense is a spindle-shaped protozoon, containing an oval trophonucleus near the center and a kineto-nucleus situated posteriorly; near the latter is found a tiny granule, the blepharoplast, which gives origin to the flagellum. The flagellum consists of its origin in the endoplasm, the undulating membrane, and the free, terminal portion. The parasite possesses an inner, granular endoplasm surrounded by ectoplasm. When fresh, infected blood is examined microscopically, it appears as a very actively wriggling parasite whose rapid movements violently agitate the red blood-cells in the preparation. The best method for demonstrating its structure is by some modification of the Romanowsky staining method.

After the discovery of Tr. gambiense, the next step was to determine its method of transmission. Years before Lewis (1877) found a somewhat similar trypanosome (Tr. lewisi) existing as a harmless inhabitant of the blood of rats and in 1880 Evans found Tr. evansi in the blood of animals in India dying from "surra." These antecedent discoveries were of great value to Sir David Bruce who, in 1894, during an investigation of "nagana" or fly disease in Zululand, found a trypanosome (Tr. brucei) in the blood of infected animals and at the same time proved that the disease was disseminated by Glossina morsitans, a species of tsetse-fly Later, Bruce and his co-workers in Uganda showed that Glossina palpalis, another biting tsetse-fly very prevalent in sleeping-sickness areas, was the agency through which sleeping sickness is spread. Bruce at first held to the theory that the fly merely acted as a mechanical carrier of the disease but Kleine showed that the fly acts as a true host for Tr. gambiense. He demonstrated that flies which have bitten individuals affected with sleeping sickness lose their infectivity within 24 hours after they have partaken of their bloody meal and that the fly only again becomes infective after the trypanosomes have undergone certain developmental changes within its body. These changes require, on the average, about 18 days, at the expiration of which time the fly becomes permanently infective. The researches of Kleine were extended by Miss Muriel Robinson who found that in the circulation of the infected vertebrate host the Tr. gambiense undergoes a cyclical reproduction and that only during these reproductive periods does the fly become the intermediary host through biting. When the fly bites an infected individual during such a period, the trypanosomes enter the stomach of the fly and divide. After multiplying in the mid-gut, they move forward into the proventriculus, thence into the hypopharynx and thence into the salivary glands of the fly. Here they again reproduce. This developmental cycle of the trypanosomes within the body of the fly requires from 12 to 20 days. Only about 8 per cent. of the flies become permanently infective.

The life-history of the parasite within the human host is but imperfectly known. During the periods in which trypanosomes cannot be demonstrated in the peripheral blood, the researches of Salvin, Moore, Breinl, Miss Robinson and others suggest that possibly the trypanosomes may occur as "latent" bodies in the lungs, spleen and bone marrow.

Trypanosoma rhodesiense very closely resembles Tr. brucei and Tr. gambiense. It is differentiated from the latter by the different location of its trophonucleus and its somewhat different action in experimental animals. It is conveyed by Glossina morsitans, the conveyor of Tr. brucei, and within this fly it has been shown to undergo a developmental cycle. Tr. rhodesiense occurs in the waterbuck, hartebeest, wart hog and in native dogs (Kinghorn and Yorke). In laboratory animals Tr. rhodesiense is more virulent than Tr. gambiense and in man the disease caused by it is more rapidly fatal. An animal having an immunity to Tr. gambiense can be infected with Tr. rhodesiense (Castellani[2]).

Antelopes, crocodiles, etc., act as natural hosts or "reservoirs" for Tr. gambiense, while "reservoirs" for Tr. rhodesiense, as before mentioned, are the waterbuck, hartebeest, wart hog and native dog.

Bionomics of the Tsetse-fly.—There are about 10 species of the tsetse-fly. The habitat of the Glossina palpalis is the tall grass and sedge near streams, trunks of trees and vines and the leaves of shrubs and bushes along the bank. It exhibits a darker deep-brown coloration than any other species of the tsetse and its position while at rest is a distinguishing character. "It is probable that only those who have suffered from the attacks of tsetse can recognize them on the wing, but in the resting position their identification is easy. In this attitude they can be separated from all other blood-sucking diptera, especially from those belonging to the genera Stomoxys and Hematopota, which are most likely to be mistaken for them, by the fact that the brownish wings lie closed flat over one another down the back, like the blades of a pair of scissors, while the proboscis (i.e., the proboscis ensheathed in the palpi) projects horizontally in front of the head" (Austen[5]).

Symptomatology.—According to Beck the course in children is similar to that in adults. Swelling of the eyelids, anxious expression of the face, and choreiform movements are prominent features.

The course of the disease, essentially a chronic one, may be divided into three stages.

Incubation Stage.—The duration of the incubation period in man is not certainly known. Apparently it is very variable, ranging from two to three weeks in some cases, to five to six years in others. During this time, although infected, the patient may show no signs of the disease. The incubation period in monkeys, experimentally infected, in which the disease is chronic and invariably fatal, is from 10 to 20 days. Castellani believes that in most human cases the incubation period is from two to three weeks.

Glandular Stage (Stage of Trypanosome Fever).—This, the second stage, is characterized by the presence of trypanosomes in the blood and lymphatics. Polyadenitis, especially an enlargement of the posterior cervical glands, is the most typical and most constant symptom.

Frequently, enlarged inguinal or femoral glands can be made out, of more or less soft consistency and varying in size from that of a pea to that of a bean. Bruce considers enlarged glands to be an early and constant feature of the disease, in fact the only constant feature of the second stage. Trypanosomes can be readily demonstrated in the gland juice.

Fever is an inconstant symptom and, when present, follows an irregular course. Bruce[1] says: "In the early stage, when the trypanosomes are found in the blood and before symptoms of sleeping sickness appear, the temperature remains normal." An intermittent fever may, in some cases, characterize the onset, persisting for about a week, later to recur at indefinite periods. An erythematous eruption, circinate, solid or rubeloid may occur, but is by no means constant. In a native, the eruption easily might be overlooked.

Frequently, during this stage, the patient may exhibit no sign of his mortal ailment other than the presence of enlarged lymphatic glands. He may claim to feel well and often pursues his work as usual. However, Bruce[1] makes the very interesting observation that the natives of Uganda recognize the enlargement of their cervical glands as the mark of death. In such a case, they eat up whatever live stock they may possess, or possibly can steal, realizing that sooner or later they will pass into the final stage of sleeping sickness, wherein there is no delight in gastronomic delicacies.

On the other hand, following repeated and increasingly frequent attacks of fever which may last for a week or longer, the patient may become anemic, weak and more or less emaciated. This stage may last for three months to three years or longer and then pass more or less abruptly into the third and final stage of the disease.

Cerebral Stage (Stage of Sleeping Sickness).—This, the final stage, is evidenced by the invasion of the trypanosomes into the cerebrospinal fluid, from the blood and lymphatics. Because of the insidious progress of the disease, there seldom is a sharp line of demarcation between the second and third stage. The cerebral stage varies in duration from a few weeks to several months. It is characterized by an increasing apathy on the part of the patient. He becomes dull, and lethargic. He may complain of headaches and indefinite pains and in voice and action he exhibits the signs of an increasing asthenia. His gait becomes shuffling and weak, his speech slow, his voice indistinct, his hands and feet tremulous. Tremors of the tongue usually are marked. An irregular fever, often normal in the morning and rising to 101 to 102°F. in the evenings, is the rule. This stage of lethargy gradually sinks into one of coma, in which the patient dies. Two to three weeks before death, the temperature permanently becomes subnormal, sinking six to seven degrees below the normal.

According to both Bruce and Castellani, lethargy and not true sleep, characterize the third stage. Bruce also brands as false the oft-repeated assertion that emaciation is one of the characteristic features of the disease. He says[1] "The body is well-nourished, and this is the rule, even up to the time of death, if the patients are well nursed and fed."

Pathology.—Gross Pathology.—In uncomplicated trypanosomiasis, the only distinctive macroscopic feature found at post-mortem is the general enlargement of the lymphatic glands and certain pathological changes in the central nervous system. The lymphatic glands, both superficial and deep, exhibit enlargement, varying from the size of a pea to that of a bean or larger. Enlargement of the glands of the neck and groin is constant. The submaxillary glands, those of the anterior and posterior triangles, the bronchial, mesenteric and the glands in the inguinal and femoral region may be enlarged and swollen. The lungs are congested, the heart muscle flabby, the liver fatty and the kidney congested.

On opening the spinal canal, an increased amount of cerebrospinal fluid is noted. The cord is often congested. On opening the calvarium, an increase in fluid is noted. The dura mater is unadherent, the surface vessels of the brain are injected, the convolutions of the brain flattened and the brain has a "ground-glass" appearance, due to the presence of cloudy subarachnoid fluid in the sulci. On section, the lateral ventricles are found to be dilated with fluid.

As in many chronic diseases, evidences of intercurrent or secondary infections, whether bacteriological or with higher parasites, are not uncommonly found at autopsy. A pneumonia and, in malarial districts, an enlarged spleen, are frequent findings. Suppurative or caseous lymphatic glands and a purulent meningitis are found with more or less frequency, but these spell a secondary bacterial invasion.

In summary, the gross changes observable at autopsy are general enlargement of the lymphatic glands and an increase in the amount of cerebrospinal fluid, with changes in the central nervous system referable to such an increase of fluid.

Microscopic Pathology.—To Mott[6] is due much of our knowledge of the histopathology of sleeping sickness. Essentially, the disease is characterized by a lymphocytic proliferation and accumulation, especially in those organs rich in lymphatics. The pia arachnoid, especially of the medulla and base of the brain shows a marked infiltration with mononuclear leukocytes, the inflammation extending along the blood-vessels and into the perivascular spaces and septa of the brain and cord. The blood shows an increase in lymphocytes and the lymphatic glands are enlarged and exhibit marked lymphocytic proliferation. As pointed out by Manson[7] and others, the extensive small cell infiltration of the perivascular connective tissue of the brain is very similar to that occurring in general paralysis of the insane.

Sleeping sickness being essentially a lymphatic disease, characterized by a proliferation and accumulation of lymphocytes throughout the body (possibly brought about by a toxin elaborated by the trypanosomes) the symptoms of the cerebral stage have been explained by

Bruce and others on the basis of a mechanical plugging of the perivascular spaces, obstructing the circulation of the brain and cord, thereby giving rise to degenerative changes in the nerve cells. The production of a toxin or toxins by Tr. gambiense has not been demonstrated, though it has been postulated by Mott and others to explain the lethargy of the cerebral stage of sleeping sickness.

Diagnosis.—In no stage of the disease is an absolute diagnosis warranted unless trypanosomes are found in the lymphatic glands, blood, or cerebrospinal fluid. In the earlier stages of the disease, a history of residence in the sleeping sickness area, enlargement of the lymphatic glands, perhaps irregular fever and the presence of a lymphocytosis should strongly suggest a search for Tr. gambiense. In the last stage, the lethargy, coupled with tremors, irregular fever and rapid pulse, the slow speech and uncertain gait, presents a more characteristic picture.

Microscopic Diagnosis.—**Lymphatic Glands.**—The demonstration by Greig that trypanosomes are present in the swollen lymphatic glands has been of immense value in diagnosis, especially in the early stages of sleeping sickness when enlargement of the lymphatic glands may be the earliest and sometimes the only evidence of the disease. By means of a sterile hypodermic syringe, several drops of gland juice are drawn off from one of the swollen glands and examined microscopically. Dutton and Todd[8] thus summarize their conclusions.

1. "The examination of glandular fluid, though not infallible, is a very efficient means of detecting the trypanosomes."

2. Because of its simplicity, gland puncture will be found a very useful routine diagnostic method. Only twice during the examination of a series of 30 cases of trypanosomiasis did a single examination of gland fluid fail to demonstrate the parasite which other methods of examination showed to be present. On seven occasions (in 26 cases) it was successful when centrifugalizing the blood had failed, and it was thrice positive (16 cases) where examinations of the cerebrospinal fluid had given negative results."

Cerebrospinal Fluid.—In the later stages of sleeping sickness, as shown by Castellani, Bruce, Broden and others, Tr. gambiense can be demonstrated in the cerebrospinal fluid, but, as Bruce[1] points out, "it is only when the symptoms of sleeping sickness have appeared that they are found with any frequency." In the final stage of the disease, the cerebrospinal fluid often is slightly turbid and in the sediment obtained by centrifugalization trypanosomes almost constantly are present.

Peripheral Blood.—The blood is best examined for the parasite by drawing 5 to 10 c.c. of blood into citrate solution, centrifugalizing and washing three times, each centrifugalization being for 15 minutes, and then examining the sediment microscopically.

Prognosis.—The disease claims a mortality of 100 per cent. The best that can be hoped for is an amelioration of the symptoms and a prolongation of life. The disease caused by Tr. rhodesiense runs a more acute course than that caused by Tr. gambiense. Differences in virulence of different strains of Tr. gambiense have been described. Laveran and Mesnil claim the disease in Uganda and the French Congo runs a more rapid course than in the Gambia. Greig and Gray believe that a small percentage of natives harboring the parasite acquire immunity to it. This is still a mooted question.

Treatment.—Arsenic, in the form of atoxyl ($NH_2C_6H_4AsOONa$) combined with antimonium in the form of Plimmers salt or tartar emetic, is considered by Castellani to be the best method of treatment, an atoxyl injection (3 gr.) being given intramuscularly every three days (as recommended by Manson) and Plimmers salt (Sodio-tartrate of antimony) administered daily—2 gr. dissolved in 2 pt. of water and administered by mouth or by rectum. If tartar emetic be used, intravenous injections of 1:100 or 1:1000 solutions, 5 to 10 c.c. per injection, is recommended, injections being given monthly on 10 consecutive days. The basis of the combined therapy was laid by the discovery of Ehrlich and his pupils that Tr. gambiense may acquire more or less marked resistance to the trypanosomicidal action of arsenic after long use of this drug alone.

In addition to antimony and atoxyl, orpiment (an inorganic salt of arsenic), mercurials, trypan red and trypan blue, parafuchsin and other dyes have been recommended.

Perhaps it has been well said that the great physician is Death.

Prophylaxis.—Intelligent prophylaxis must be based on the knowledge that sleeping sickness is spread by the tsetse-fly, that the disease follows lines of intercommunication and that certain big game in Africa serve as reservoirs for the infection. At present, avoidance of the sleeping sickness area is the only sure mode of escape. The destruction of the carrier, the tsetse-fly appears, at present, to be an almost insurmountable task, though clearing of the bush and other breeding places of the fly is being attempted more or less successfully in certain areas. Osler[9] says: "The hope appears to be in the extermination of the animals upon which the Glossina palpalis feeds." Isolation of infected natives and forbiddance of passage of natives into infected areas are measures theoretically advisable, but, at present, exceedingly difficult of accomplishment. Tropical Africa is confronted with a stupendous task in her efforts to control the ravages and spread of sleeping sickness.

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SOUTH AMERICAN TRYPANOSOMIASIS

Synonym.—Infantile Cachexia.

Definition.—South American Trypanosomiasis is a specific infectious disease, occurring especially in children in the state of Minas, Brazil, characterized by an acute or chronic stage, and caused by Trypanosoma cruzi.

History.—The discovery and naming of T. cruzi is due to Dr. Carlos Chagas, of the Oswaldo Cruz Institute in Rio de Janiero. In 1909 he found the trypanosoma in the intestine of Lamus (Traitoma) megistus, a voracious bug which his investigations showed was present in great numbers in the houses of the poorer inhabitants of the state of Minas. This bug especially attacks children while asleep at night, inflicting its bite most frequently upon the face. From this latter fact, the native name for the Lamus is "barbeiro" or barber. The bite is painful, the insect "barber" emulating certain of his human confréres in leaving upon the face some tangible evidence of his excursions. Chaga's first demonstration of a trypanosome, identical with T. cruzi, in the blood of the human, was made on a two year old child in the state of Minas, who was ill with an irregular fever, marked anemia and enlargement of various lymphatic glands. During the years 1909–1912, Chagas succeeded in cultivating the trypanosome, worked out its life history, found the apparent reservoir to be an armadillo, Dosypus novemcinctus, and worked out the clinical and pathological aspects of the disease.

Etiology.—**Geographical Distribution.**—The disease is confined to the poorer inhabitants of the state of Minas, Brazil, among whom, apparently, the disease is universal.

Age.—South American Trypanosomiasis is a disease of children, hence its name "infantile cachexia." All of the poorer children apparently contract the disease. Some die of the acute form, some pass into the chronic stage and others recover.

Exciting Cause.—The exciting cause of infantile cachexia is T. cruzi, a trypanosome characterized by an unusually large, oval kineto-nucleus situated posteriorly in the cell and possessing a chromatin appendage. This form of the trypanosome occurs free in the blood, as does also another form described by Chagas, which is broader and possesses a rounder and smaller kineto-nucleus.

In addition to the above-mentioned forms of T. cruzi which occur free in the blood, intracorpuscular forms of the parasite have been described. These are said to lie either partially or completely within the red blood-cell.

T. cruzi is not known to undergo longitudinal division within the body. Schizogony occurs in the lungs, with the formation of merozoites, which enter the erythrocytes, develop a flagellum, and again assume the typical trypanosome form.

Sporogony occurs within the body of the insect host, Lamus megistus, and, according to Chagas, both an asexual and a sexual form of development occurs. The final stage of the sexual process of development results in the formation of long, slim trypaniform forms which are found in the salivary glands of the bug. These are the infective forms, passing into the vertebrate host in the act of biting.

Cultivation.—T. cruzi is somewhat remarkable in the case with which it can be cultivated on artificial media. The Novy-McNeal blood-agar is the medium recommended and therein a development cycle occurs which closely resembles that which takes place naturally within the body of Lamus megistus.

Pathogenicity.—After an infective meal, the larvæ of Lamus lose their infectivity for vertebrates within two to three days, becoming again infective for man after 8 to 10 days. Monkeys are capable of infection. When so infected, Lamus becomes infective when allowed to feed upon them. Rather curiously, according to Chagas, the infected guinea pig, is not infective for Lamus.

Pathology.—**Gross Pathology.**—An autopsy on a patient who has died during an acute phase of the disease reveals an enlarged, fattily-degenerated liver, an enlarged and hyperemic spleen and mesenteric glands, and some serous effusion within the abdominal cavity. On opening the chest there is found a serous effusion in the pleural and pericardial sacs, with hemorrhagic pericarditis and an enlarged heart exhibiting marked myocarditis. The mediastinal lymph glands are enlarged. In the neck, the thyroid and cervical lymph glands show enlargement. As regards the nervous system, the cerebrospinal fluid is increased in amount, the dura is congested and there is present a leptomeningitis and encephalo-meningitis.

Microscopic Anatomy.—Upon entrance into the vertebrate body, the trypanosomes exhibit a special predilection for the muscles, especially the muscles of the extremities and back. In the skeletal muscles, as well as in the heart muscle, the parasites grow and divide within the muscle fibers, destroying the sarcoplasm of the cells and, by their multiplication, giving rise to parasitic cysts. When these cysts rupture, the trypanosomes escape into the surrounding tissue and gain entrance to the blood stream, whereupon general symptoms of the disease manifest themselves. In other tissues and organs of the body, as well as the muscular system, the parasites enter the body cells, give rise to cysts, and on the rupture of these cysts the escape of the trypanosomes sets up local inflammatory changes.

In the central nervous system, the neuroglia cells are attacked, converted into cysts, and when the cysts rupture, a local inflammatory reaction occurs. A similar invasion of the parasites has been demonstrated in the thyroid gland, the kidneys, the hypophysis and the suprarenal glands.

Symptomatology.—Two varieties of the disease are noted by Chagas—the acute and the chronic.

Acute Form.—In a young child the disease is ushered in by high fever, thyroid gland enlargement, swelling of various groups of lymphatic glands, an enlarged and painful spleen, hepatic enlargement, and edema of the face. The attack subsides, to be followed by subsequent periodic attacks, during which the child recovers, dies, or passes into the chronic stage of the disease. Trypanosomes are found in the circulating blood only during the pyrexial attacks.[2]

Chronic Stage.—Chagas classifies the chronic stage of the disease into five types: (1) The pseudo-myxedematous; (2) the myxedematous; (3) the cardiac; (4) the nervous; (5) the chronic form with acute or subacute exacerbations. This latter form is the cause of much mortality and is distinguished from the acute type by the long duration of the illness and the scarcity of trypanosomes in the circulating blood.

Sequelæ.—Infantilism associated with hypothyroidism is believed by Chagas to be a not infrequent result of the chronic form of the disease.

Diagnosis.—In a febrile period of the acute stage, a positive diagnosis can be made by finding T. cruzi in the peripheral blood. This is the only method by which the condition can be absolutely differentiated from hook-worm, which a personal communication from Dr. Hackett, Director of the Rockefeller International Health Commission for Brazil, assures the author is exceedingly prevalent among the poorer inhabitants of the state of Minas, and malaria, the two diseases with which infantile cachexia is most easily confounded.

Prognosis.—In the acute form, in the chronic form with acute exacerbations and in the marked cardiac form, the prognosis is very serious.

Treatment.—The treatment is that for African trypanosomiasis, plus therapy directed to an alleviation of the hypo-thyroidism.

Prophylaxis.—As the lamus is the carrier of the disease, prevention of its biting man and eventual eradication of the bug are the prophylactic measures to be striven for.

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CHAPTER CXXXI

MALTA FEVER

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Synonyms.—Undulant fever, Mediterranean fever, Gibraltar fever, Neopolitan fever, etc.

Definition.—Malta fever is a febrile disease, usually with undulatory relapses and with enlarged spleen, caused by Micrococcus melitensis and chiefly spread through the agency of infected goat's milk.

History.—According to Castellani[1] the first accurate account of the clinical aspects of the disease and its post-mortem appearances was given by Marston in 1859. In 1887, Bruce[2] reported the discovery of the causative microörganism and its cultivation from the spleen of five patients in Malta dead with the disease. In 1897, Wright and Smith[3] reported that patients infected with Micrococcus melitensis possessed in their blood specific agglutinins. The exhaustive study made by the British Mediterranean Fever Commission (1905–1907) established the fact that the ingestion of infected goat's milk is the important mode of infection for man. They showed that about 50 per cent. of the goats in Malta are carriers of the disease; that about 10 per cent. of such animals excrete the Micrococcus melitensis in their milk; that such infected milk, when fed to monkeys, gave rise to the disease; that in about 10 per cent. of the cases the microörganism is excreted in the urine. The recommendation of the Commission that the use of goat's milk be prohibited resulted in a reduction of the disease among British troops stationed in Malta of from 643 cases in 1905 to one case in 1910 and in a reduction of more than 95 per cent. in the case incidence on the Island (Eyre[4]).

Distribution.—Malta fever, or perhaps, more properly, undulant fever, has a wide distribution, occurring not only in the islands of the Mediterranean, but in many tropical and subtropical countries as well. The disease has been reported from Italy, Greece, Turkey, Spain, Portugal, France, Arabia, India, East and South Africa, China, Ceylon, the Philippines, West India, North and South America.

The first evidence of the endemicity of Malta fever in the United States was given by Gentry and Ferenbaugh.[5] They found it to exist in endemic form in Edwards County, Texas, the principal goat-raising section of the state. Their reports detail clinical and laboratory findings on 12 human cases. Also, out of 151 goats examined, the blood of about 20 per cent. gave positive agglutination with M. melitensis at 1:20 dilution. One of their conclusions is: "Although typhoid fever is prevalent throughout these sections, we believe that careful serum tests will show that a large proportion of the cases diagnosed as atypical typhoid fever will be found to be suffering from infection with M. melitensis."

Age Incidence.—It may be said that no age is immune to Malta Fever. Williams mentions a case of intrauterine transmission of Malta Fever, and Haushalter reports a three year old child who died after developing meningeal symptoms due to the micrococcus melitensis.

Etiology.—Micrococcus melitensis.

Morphology.—The exciting cause of Malta fever is ordinarily described as a minute, oval coccus. Bruce,[2] in his original communication and elsewhere, considered it to be a micrococcus. Jordan,[6] following Babes and others, describes it as "a very small coccus-like bacillus, about 0.5μ or less in diameter, usually occurring singly or in pairs, though in cultures short chains are found." It is aërobic, non-motile, stains readily with ordinary anilin dyes and is Gram-negative.

Cultivation.—It is characterized by its slow, rather non-characteristic growth upon artificial media, even at 37°C., its optimum growth temperature. Upon plain agar, at 37°C., minute, transparent colonies, with a slightly yellowish center, develop in three to four days. In broth a slight turbidity occurs after four to six days incubation at 37°C. Gelatine is not liquefied, no acid is produced from dextrose, and no indol is formed. Milk, especially goat's milk, is a good medium for growth and develops a progressively deeper alkaline reaction. The micrococcus grows best upon media slightly alkaline to phenolphthalein. The addition of 1 per cent. nutrose is said to favor its growth.

Vitality.—In a dried state, it retains its viability for a considerable length of time, withstanding drying in dust for 60 to 80 days. In markedly alkaline urine it has remained viable for at least six days (Bruce[7]).

Habitat in the Body.—*Portals of Entry.*—The usual avenue of infection for man is the alimentary canal and practically the only method of infection worth considering is the ingestion of infected goat's milk. Although Kennedy, of the Mediterranean Fever Commission, demonstrated that living micrococci are excreted in the urine of 10 per cent. of the patients and although the microörganisms probably escape in the feces, human infection from these sources has never been proven.

Portals of Exit.—The urine of patients may contain M. melitensis in enormous numbers, although, as a rule, the microörganisms are not numerous. An important fact is that patients may excrete micrococci in the urine for as long as two years after convalescence (Bruce[7]).

In artificially inoculated rabbits, Eyre[4] found living micrococci throughout the entire alimentary tract. The presence, however, of M. melitensis in the feces of human cases is still largely unproven.

The third method of escape from the body is in the milk. In 1906, Bruce reported the finding of micrococci in the milk of two out of three women suffering from Malta fever.

Infection from goat to goat may be kept up, according to Brumpt, by the fact that goats will drink human and goat urine.

Incubation.—The exact period of incubation is not known. It appears to vary from 6 to 15 days.

Immunity.—It is not definitely known whether one attack of Malta fever confers immunity. Specific agglutinins for M. melitensis may persist in the blood for from 4 to 10 years after an attack (Castellani[1]).

Pathology.—The disease is a septicemia, the organisms gaining entrance to the blood through the intestinal mucosa. The most marked feature is the congestion and enlargement of the spleen. In this, it may resemble typhoid but Bruce[7] points out that, in marked contrast to typhoid fever, the mesenteric glands show only moderate enlargement.

The spleen may weigh from 10 to 44 oz. (Castellani[1]), averaging about 20 oz. The liver, kidneys and lungs show congestion and in the lungs patches of consolidation may occur.

Symptomatology.—The onset is gradual, with symptoms commonly met with in other fevers, such as malaise, headache, chilliness as prodromata. By the end of four to five days the patient exhibits an evening temperature of about 103 degrees. The spleen enlarges and becomes tender within a week or 10 days after symptoms of the disease manifest themselves. Constipation is said to be a marked symptom. Swollen and painful joints are common.

The disease is characterized by marked irregularity of fever and a decided tendency to relapses. After several weeks, during which the temperature ranges from 103 to 105 degrees, the temperature may fall to normal. Then, in several days, a relapse occurs and relapses and intermissions may alternate for months.

The average duration of the illness is variously estimated at from 90 to 120 days.

Diagnosis.—The surest method of diagnosis is by means of blood cultures. The micrococcus has been cultivated from the circulating blood in 82 per cent. of the cases thus examined (Jordan[6]). Bruce was the first to obtain cultures of M. melitensis by means of spleen puncture during life and from the spleen post-mortem.

The agglutination test is a valuable aid. For this test, Castellani[1] recommends a dilution of 1:80 with a time limit of 30 minutes. Bruce[7] says: "A complete sedimentation is never obtained at 1 in 20 unless the blood is from a patient with Malta fever."

The blood shows a secondary anemia, with reduced hemoglobin and a red-cell count 20 to 40 per cent below normal. The polymorphonuclears are decreased in number and the mononuclears are increased, even to as high as 80 per cent. in some cases.

Prognosis.—Ordinarily, the prognosis is good. Bruce[7] places the mortality at from 2 to 3 per cent. Death usually occurs within the first two weeks of the disease.

Treatment.—The treatment is entirely symptomatic and is similar to that for typhoid fever. Hydrotherapy is valuable in controlling the fever. Vaccin therapy has been attended with but little success.

Prophylaxis.—Prophylaxis is spelled in four words "avoidance of goat's milk!"

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CHAPTER CXXXII

KALA-AZAR IN CHILDREN (LEISHMANIA)

By Fritz B. Talbot, M.D.

Boston, Mass.

Kala-azar, also known as black fever, tropical splenomegaly, dum-dum fever, and cachectic fever, is a disease characterized by marked enlargement of the spleen, emaciation, cachexia, and irregular fever, and caused by a protozoal organism known as the "Leishman-Donovan body." The disease is common in India, where it was found by the English in 1869, and is found chiefly among adults. It also occurs in China, Turkestan, Arabia, Egypt, Tripoli, Algiers, Italy, Greece, Malta, Sicily, Spain, and Portugal. It is said that in Assam over 26 per cent. of the population is infected before the tenth year, and 50 per cent. or more before the twentieth year. The cases reported from the countries bordering on the Mediterranean, chiefly found among young children, were not numerous until recent years, when large series of cases have been reported.

Etiology.—The earlier investigators thought kala-azar identical with malaria In May, 1903, however, Leishman discovered the parasitic bodies, which bear his name, and distinguished the disease from malaria. These parasites were found by him, and also by Donovan, in the spleen, liver, and epithelium of the blood-vessels. They may also be present in the bone marrow, mesenteric glands, kidneys, brain, peripheral lymph-nodes, and intestinal ulcers. In 75 per cent. of the advanced cases reported by Donovan and Patton,[1] they were found in the peripheral blood. In 1905 Pianese[2] found the parasites in certain splenic anemias of infants at Naples. In 1907, Ch. Nicolle[3] found the disease in Tunis, cultivated the parasite, and reproduced the disease in the monkey and dog. In 1908, in collaboration with Comte, Nicolle[4] demonstrated the existence of canine Leishmanosis, which has the same geographical distribution as infantile kala-azar. Because of the localization of the disease, the susceptibility of the dog, and its coincidence in human beings, he concluded that the disease in the human and in the dog was identical. He believed, also, that kala-azar was carried from dog to dog, and from dog to infant, by fleas. Up to date, however, it has not been possible to transmit the Indian form of the disease to animals. Organisms from the spleen of patients suffering from kala-azar were cultivated upon agar, streaked with fresh human blood, by Rogers. The parasite in tissue is round, or oval, with a granular protoplasm, resembling, at first sight, blood platelets, and measures 2 to 3.5 μ in diameter. The nucleus is oval and located in the center of the parasite, during the resting stage, and at the periphery, prior to division. A small rod, perpendicular to or at a tangent with the nucleus, is present at one side and stains more deeply than the nucleus. The parasites are usually intra-cellular, but may be extra-cellular. When cultivated artificially, they enlarge rapidly attaining the size of 7 to 9 μ. They become flagellated and pyriform in shape. They have not, however, an undulating membrane as do the trypanosomes. The flagellated forms grow to 12 or 20 μ in length, and multiply by longitudinal fission, during which they sometimes resemble spirillæ.

Transmission.—Transmission of the disease is by close contact with infected persons or animals. There is much evidence that either the flea or bed bug are the hosts through which this transmission is accomplished.

Incubation Period.—This is indefinite and varies from three weeks to several months, though according to Whipman,[6] an incubation period of less than 10 days has been known.

Symptoms.—In the neighborhood of the Mediterranean, kala-azar is found almost exclusively in infants from six months to three years of age. Unlike the adult form found in India, the onset is usually insidious, although it may commence with chills, high fever, and vomiting. The fever is intermittent or remittent, the temperature chart often showing two remissions in the course of 24 hours. The fever lasts six weeks or longer, and then declines. During the period of pyrexia, the spleen and liver enlarge. This may be followed by periods of lower temperature, during which the spleen and liver decrease in size, subsequently enlarging with the return of high fever. The periods of high fever may recur for months. As the disease progresses, anemia becomes pronounced, the infant loses weight, interest in his surroundings, and ceases to play. Loss of weight may progress to cachexia. Often the enlarged abdomen, due to the great size of the liver and spleen, in contrast with the general emaciation, is the prominent symptom. The abdominal veins are enlarged, and there may be white, soft edema of the eyelids, hands and feet, which resembles that in Bright's disease. Ascites may be present. There is diminished appetite and often diarrhea. In very pronounced cases there may be hemorrhages of the gums, epistaxis, purpura, and at times bullous eruptions. In many cases the skin acquires a peculiar earthy-gray color, and there may be jaundice. The hair is dull and brittle, and may fall out. The glands in all parts of the body tend to enlarge.

Blood Picture.—Leukopenia is characteristic and may vary from 6000 whites to 2400. Baths given to reduce fever may convert a severe leukopenia into a temporary slight leukocytosis. In advanced cases the hemoglobin is 35 to 40 per cent., and the red count about 2,500,000 per cubic millimeter. The stained smear shows a secondary anemia, with occasional normoblasts. There is a marked increase of lymphocytes, especially large mononuclears, with a corresponding reduction of polynuclear neutrophiles. Blood platelets are increased in number. The coagulation time of the blood may be slightly decreased. Occasionally, the parasites may be demonstrated in the blood, but this is very unusual in infants and young children.

Diagnosis.—Diagnosis lies between malaria, Banti's disease, lymphatic leukemia, Malta fever, typhoid, paratyphoid, and pseudo-kala-azar. The type of fever, with remissions and intermissions, associated with greatly enlarged spleen, and an enlarged liver, is said to be characteristic. The diagnosis, however, can only be made by splenic or liver puncture, and demonstration of the parasite in the smears of the organ or its blood. The organism may be more easily demonstrated in smears of the spleen juice than from the liver, but great care should be taken in puncturing the spleen, because of the danger of hemorrhage. For this reason many prefer to puncture the liver, others examine the bone marrow of a rib or the tibia. In a case on the writer's service in the Children's Ward of the Massachusetts General Hospital, a culture was grown from an excised inguinal gland.

Prognosis.—This is usually fatal, 96 per cent. dying. These figures have been recently modified, however, by Rogers,[8] who reports a large number of cures by the administration of tartar emetic.

Treatment.—The treatment, until recently, is said to be unsatisfactory. Intramuscular injections of atoxyl have been recommended as well as quinine and salvarsan, but the results have not warranted their use. Vianna, in 1912,[9] first gave tartar emetic therapeutically and his results have been enthusiastically followed up by Rogers and others, who report a large proportion of cures with its use. Rogers[10] gives a 2 per cent. solution of tartar emetic intravenously commencing with 1 c.c. and increasing, in children, if possible, up to 5 c.c. Repeated doses are given until the fever stops. The writer's case was cured by this means. During one stage of the treatment the child received so much tartar emetic that she was unable to eat because of her nausea. Improvement from surgical measures, such as splenectomy, is, as a rule, only temporary.

CHAPTER CXXXIII

YELLOW FEVER OF CHILDREN

By H. R. Carter, M.D.

Baltimore, Md.

Yellow fever in children has been very little studied from a clinical standpoint. Its importance as a factor in epidemiology however, has been recognized in children and has formed the very vortex of conflicting opinion, out of which has come a knowledge of its epidemiological relations that has furnished us with the key by which much disputed questions of high importance have been answered. It does not seem to me that a discussion of the general question of yellow fever is in place here. For a knowledge of the general subject, the reader is referred to systematic treatises. The bulk of this article then will be devoted to its epidemiological relations. In dealing with its clinical history, only the *differences* in its manifestations in children as compared with those in adults will be given; while in the epidemiology, such points will be discussed as seem especially dependent on the existence of the fever in children.

The disease is contracted by a child in the same way as by an adult —through the bite of a mosquito, Aëdes (Stegomyia) egypti herself infected by having fed on a man sick of yellow fever. *In nature* it can be contracted in no other way. Consequently there is nothing peculiar in the sanitary management of yellow fever in children, once it is recognized, other than that required for adults.

There seems no reason why the period of incubation in the child should be different from that in the adult. In the few cases observed with a single definite exposure (at Orwood and Taylor, Miss.) it was within the same limits—not more than six nor less than three days. No experimental cases among children are on record.

The question of whether children are immune to yellow fever is barred by the title of this article. Whether children born in a special locality or of immune parents are so immune will be taken up later.

The general signs and symptoms of the disease evolve similarly in the child as in the adult. The disease in children, especially those under five years, is very much milder, even ephemeral. Probably it would be more accurate to say that the proportion of mild and very mild cases is much greater among children; because grave and fatal cases do occur among them as among adults. Naturally, in these very mild cases the distinctive signs and symptoms on which the diagnosis is based are less well marked and, without the use of laboratory methods the diagnosis of this disease in children, especially in young children, is difficult and in many cases impossible.*

All cases, however, are not mild. In years when the yellow fever is epidemic the mortality of white children is markedly increased. Yet in places in which yellow fever is endemic, even these fatal, and hence necessarily severe cases, if they occur in native children, are rarely recognized. This, however, is due to erroneous nosological theory rather than to the lack of the yellow fever characteristics. Doctor Finlay of Havana told me that he was not sure that he had ever seen a case of yellow fever in a Cuban (Havana?) child! Yet "Borras," a fever of children with black vomit, was well known in Havana! During the yellow fever in Panama in the early days of the Canal, I saw only one case in a child, the daughter of the Peruvian Minister, and I saw not only *all* cases reported as yellow fever, but all those suspiciously resembling it. Yet there were many children in Panama and a fair amount of yellow fever. We will recur to this later.

Clinical History.—The clinical observations of yellow fever in children available to me are few. I think there are few on record. The writer has seen a good number of cases—only a few of which, however, he has studied. In them the history of the disease for young children, not over five, was about as follows:

The child is taken sick suddenly with a fever. Unlike malarial fever, vomiting and acute pain in the abdomen are rare as initial symptoms. There is restlessness and fretfulness rather than languor. The child shows that it has headache by frowning, hiding from light, etc. Some complain of the pains in the back and limbs, frequently so distressing in older people, but I found much less complaint of this pain in the yellow fever cases than among the children who were suffering with dengue in Porto Rico in 1915. Nevertheless these pains are present and at times are severe. One case, a fatal one, showed the classical picture of the "coup de barre." From the history, a number of these children's cases began with sore throat, so that they were thought to be pharyngitis or tonsillitis. This, however, occurs occasionally with adults also, probably more frequently than is generally known. It seems, however, to be more common in children.

The facies of the early stage is as well marked in children as in adults, the active congestion of skin and mucous membranes which produces these facies prevailing similarly in the young and old. The facies are very like those at the beginning of measles, prior to the eruption, except that they have less lachrymation and no coryza.

Guitéras, a most careful and experienced diagnostician, states that the conjunctiva even at this stage shows a tinge of yellow. I have not always looked for it and not always found it when I looked, yet as a differential characteristic between children and adults it *has* been more commonly found in them at this stage than in adults. My experience does not verify this early jaundice as a differential sign in men, doubtless because my adult patients were mainly men leading an out-door life in the sun and normally showing a yellow conjunctiva, while the children did not. On the whole, I think it is common in children and of value as a diagnostic sign. I have never myself seen a case in a young child, under five, in which I could have *positively* made the diagnosis at this stage independently of environment. It is only fair to say that I have seen but a few such cases to study them. I have heard, however, of cases in young children described by other men which even at this stage would have *compelled* diagnosis. Some of them, 8 and 10 years old, like adults have shown quite well-marked characteristics at this stage.

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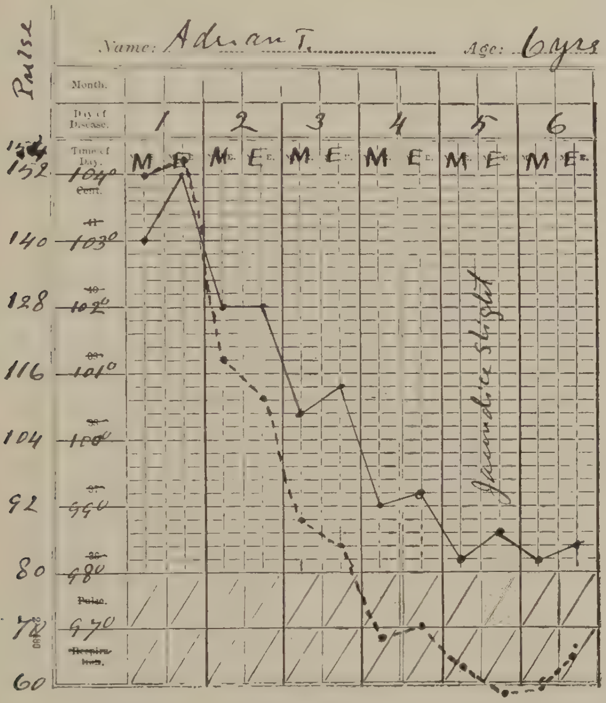
* The reaction of the serum of the suspected patient with cultures of *Leptospira icteroides* is considered to be determinative of the diagnosis by a very considerable number of men familiar with the subject.

This stage of active congestion then is very suggestive and, is the disease be only a question between yellow fever and malaria, if frequently sufficient to make the diagnosis. This, indeed, is true for young children also. The stage of stasis ("stage of calm") is usually well marked in both young and older children, with the same signs and symptoms that an adult presents. The dusky congested face, the "tâche" slowly fading and quite generally showing the skin yellow under pressure, and above all, the placid, contented languor of this stage are the same as in adults.

Occasionally the older children showed gums bleeding on pressure, while girls of even as young as 10 years very generally bleed from the genitalia. Jaundice seems more common, but less profound, than in adults. The first probably due to being easier to detect in children, especially in the conjunctiva; the latter because the attacks averaged much lighter.

The temperature was frequently high on the first day of the attack —103 to 104 degrees and even higher, but in most cases fell rapidly. Most cases after the first day ran not over 102 degrees. A temperature continuing over 103 degrees was of bad augury. The pulse, as in adults, was too high for the temperature in the beginning and then followed Faget's law, falling disproportionately more than the temperature. I think this law was as much observed with children as with adults—allowing for the greater mobility of the pulse rate. I have not seen in children the extremely slow pulses of 30 and 40 per minute sometimes found in adults. The essence of a Faget pulse is not that the pulse falls, but that the portion of the ordinate between the lines of pulse and temperature should *increase* constantly, the pulse falling away from the temperature. It is, in adults, less generally observed in severe cases than in moderate and light ones. A rise in pulse rate is of less prognostic import than with adults. I would not like to see it persist, however.

In the stage of stasis, usually in the third or fourth day, albumen showed, if it showed at all. It occurred, certainly, less frequently in children than in adults, was less in amount and was found to be more evanescent. This difference existed even in cases with equal temperature reactions. Quite frequently no albumen was found, even when there was an opportunity for a careful examination of the urine for the first four days. Suppression of urine is also rarer among children, even with cases whose other symptoms are equally grave. Indeed, I have never seen this suppression, although it has been reported by others. The functional kidney disturbance is less in the child than in the adult.



——— Temp.
. . . . Pulse

CHART No. 1.—Case of yellow fever showing Paget's sign. High temperature on first day. Rapid fall on second and third. The normal pulse for this age is taken as 86 and the normal rise for 1°F. of temperature as 12 heats probably a little too small for six years old.

On the other hand, slight black vomit, "fly-wings," is, for the same general gravity of symptoms, more common in the child than in the adult and is much less frequently followed by full "black vomit." This last has seemed to me of less grave prognosis than with adults, but I have not seen many cases of it. Tarry stools—"black vomit stools"—occur oftener than the vomit itself. A "black vomit" in the initial period (stage of acute congestion) has been seen twice. It was believed to be due to blood swallowed from oozing in the pharynx or posterior nares. It was decidedly more commonly observed in dengue than in yellow fever. Tenderness on pressure in the epigastrium is common, as in adults. I have seen less complaint of *pain* in this region, as distinguished from tenderness, and it is a wise rule, having once elicited tenderness, to press this region no more. Death was practically always in convulsions, preceded by cerebral symptoms, delirium and coma. It seemed to be due to a pure toxemia from the infection. I have not seen it associated with suppression of urine as is not uncommon among adults. Such cases occurs, however.

Treatment.—No difference need be noted in the treatment between children and adults. If the diagnosis be made early, the specific serum of Noguchi should be used at once. Otherwise it is symptomatic in both. The child has the advantage of not being scared or anxious about his affairs—mental states which at times count heavily against the adult—nor is there, as in the adult, the same danger arising of the toxins, due to the diminished secretion from the nephropathy, being added to the toxemia from the infection. In the beginning a laxative, not a purge, is very generally accepted as good practice. Although the good resulting from such treatment is not by any means a proven thing, yet it seems reasonable that to open the bowels, which are quite generally bound in the beginning, and prevent them from being bound would lessen the possibility of toxemia from intestinal absorption being added to that of the infection. There is a largely increased excretion of indol (in adults); so intestinal absorption is probably not lacking.

Absolute rest in bed for the whole period of sickness and absolute diet (i.e., *water*) for the first few days are necessary. Care must be taken with the child not to continue this diet to the point where it causes dangerous weakness. I never saw an adult case hurt by fasting; whereas I have seen children endangered in this way. In giving food, remember that the stomach is usually *very* acid and that milk is almost sure to form large curds and induce vomiting. The water from rice, which is thick with starch, or juice from stewed oysters is better than milk. My old friend, Doctor Faget, was very fond of Ducro's Elixir. I thought it a good enough stimulant, but doubted if it had any food value. The crumbled yolk of a hard boiled egg is usually well borne. In case of doubt as to feeding or withholding food, the latter is the safer policy. It is easy to restore from weakness, hard to check black vomit. Guard from any exertion or from feeding when the temperature first falls and the child, although languid, feels comfortable—lest this period be indeed, as old Dutroulau calls it: "Le mieux de la mort." As much water should be given as can be taken without nausea. This water should always be slightly alkaline, and if desired may be carbonated. If necessary supply it by enemata. The same ingestion of water is equally, or even more, advisable for the adult.

The only autopsy made, on a child of eight years, gave the same appearances as found in the adult.

As illustrating the above: A Creole family* was seen in 1898 with Doctor Faget of New Orleans, an acute diagnostician. The parents had yellow fever in 1878 but were not sick at the time seen. There were eight children. A girl of 16 had had, from the history, a severe attack, with black vomit, but was fully recovered. A boy of 13 and a girl of 16, twin of the first one, both had well-marked but light attacks; the girl's, in fact, being extremely mild. A boy of 9 or 10 was sick, but considering him alone, the diagnosis was impossible. A boy of six was quite sick and the diagnosis positive. Two children, about four and three years, were sick as was the baby about a year old. All had fever, were fretful, "cross," and listless. Their sickness lasted only a few days. The baby was somewhat jaundiced in convalescence. It was agreed that all the children had the same disease, yellow fever, but the diagnosis of the boy of 10 and the two little children was only possible by considering the circumstances under which they became sick. The boy of 10 did not show albuminuria.

The above experience is by no means unique. In a number of instances children were believed to be suffering from yellow fever, although there was nothing characteristic in their sickness, because other members of the family were having the disease at the time. Considered by itself diagnosis in some of these children had been impossible: "A simple fever of undetermined nature" had been the record.

Prognosis.—The prognosis in children is good, varying, as with adults, with the type of the epidemic, if an epidemic exists. Yet in the same epidemic, prognosis is much better for children than for adults. In children it shows the same racial difference between blacks and whites as with adults.

While the writer is disposed to regard it as a mild disease in children, in some epidemics almost trivial, yet the *absolute* mortality of an epidemic may be large. The disease when frankly epidemic is so prevalent, attacking practically all of the susceptible people in the community, that while its proportional case-mortality may be low, yet on account of the great number of cases, its absolute mortality may well be large.

Guitéras and Chaillé show that there is always during yellow fever epidemics an excess of deaths of children—frequently very large.† The mortuary records rarely ascribe this excess to yellow fever, never all of it, yet the phenomenon of this excess is so invariable and, where race is recorded, is so unfailingly made up of the deaths of *white* children that the conclusion that it is due in its entirety to yellow fever is irresistible. Of course, the concurrent existence of epidemics of measles, whooping-cough, and such other known causes of infant mortality, would require us to modify this statement. In these cases, however, there is an increase of deaths among black children also, greater (Guitéras has shown) than among the white.

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* By Creole is meant one of Latin descent (French, Spanish or Portuguese) born in America. A Creole then is of the white race.

† Acclimatization or acquisition of immunity from yellow fever. Stanford E. Chaillé, National Board of Health Report (1880). Some observations on the natural history of epidemics of yellow fever based on the mortality statistics of the city of Key West. John Guitéras, U. S. Marine Hospital Report (1888).

The following tables are adapted from those of Chaillé, giving the deaths of children during a period when yellow fever was epidemic in New Orleans and for a similar period when it was not epidemic.

Adapted from tables of Stanford E. Chaillé of New Orleans, La. (op. cit.).
Yellow Fever Epidemic 1858 August, September and October.
1867 September and October.
1878 August, September and October.
Not epidemic, 1857–1859; 1866–1868; 1877–1879.

TABLE 1

	1857 and 1859	1858	Excess of deaths during these three months of 1858, over the average of 1857 and 1859
	Deaths during August, September and October, average for the two years	Deaths during August, September and October	
Under 1 year	409	632	223
To 5 years	623	1487	864
To 10 years	681	1665	984

Note that there was a very severe epidemic in 1853 and that therefore in 1858 there were but few of those over five years old, born in the city, but had had an attack of yellow fever. The excess of deaths between 5 and 10 years is only 120—24 for each year of life as against 173 for each year of life before five. There was considerable immigration to New Orleans during this time and, naturally, some of families with children.

TABLE 2

	1866 and 1867	1867	Excess of deaths in these two months of 1867; over the average of same months in 1866 and 1868
	Deaths during September and October, average for the two years	Deaths during September and October	
Under 1 year	263	461	198
To 5 years	409	914	505
To 10 years	475	1225	750

1867 was not considered a bad epidemic. To it is accredited 3107 deaths as against 4855 in 1858 and 4046 in 1878, and note that the excess of child mortality is less in 1867 than in 1858 or 1878.

Table 3

	1877 and 1879	1878	Excess of deaths in 1878 during these three months over the average of the same three months in 1877 and 1879	1878	
	Deaths during August, September and October, average of the two years	Deaths during August, September and October		Deaths of yellow fever reported among children	
				White	Colored
Under 1 year...	239	402	163	53	5
To 5 years.....	372	1901	1529	1025	54
To 10 years....	420	2444	2024	1404	76
10 to 15 years..	32	225	193	176	14

There had been no severe epidemic of yellow fever since 1867 so that doubtless few of the native born children under 11 years of age in 1878 had been exposed to the disease prior to this year, while many over that age had been. Note, the excess of deaths between 5 and 10 (495) is much larger than in 1858 (120), although the latter was, if anything, the severer epidemic. Note, too, the rapid fall of this excess above 10 years (probably really above 11 years), it being 193 for the five years between 10 and 15 as compared with 495 for the preceding five year period. The number of the colored population of New Orleans at this time was approximately equal to the white, probably a little smaller.

These tables are self-explanatory. In months when yellow fever prevailed there is, for every tabulated age interval of childhood, an excess of deaths over the corresponding months of adjacent years in which it did not prevail. The deduction is irresistible.

It will be seen in the table of 1878 that a number of deaths from yellow fever is recorded among children. In the records for previous years made out by Chaillé. There is no record of deaths from yellow fever among the children of New Orleans. The record of 1878 is due to the fact that prior to 1878 there existed the almost universal belief in New Orleans that yellow fever did not occur among the Creoles. Hence any diagnosis rather than yellow fever was accepted for such children. The heavy mortality among the native born in 1878 (there had been no general epidemic for 11 years, and a susceptible native population had grown up) convinced many physicians of the fallacy of this opinion, and hence the diagnosis of yellow fever was freely recorded for children this year.

It is noteworthy that in Brazil, as in New Orleans in 1878, yellow fever in native-born children was frequently recorded, while in Cuba and in other endemic centers of which I have knowledge, such records were never made. The reason for Brazil's advance in this respect is that the yellow fever in that country is of recent introduction, 1849, and thus is within the memory of the last generation of physicians. In that year the children were attacked by the disease in such large numbers that the tradition of it still lingers. The disease was readily recognized among children in those first epidemics—1849 to 1852—because (1) it was a new disease and (2) it occurred at once among the whole population of every age, *compelling* recognition among the adults and showing every grade of severity among the older and younger children. It was doubtless for the same reason that Blair recognized it so frankly in children in Guiana in 1852, this being the first frank epidemic in Guiana for many years, and hence universally prevalent.

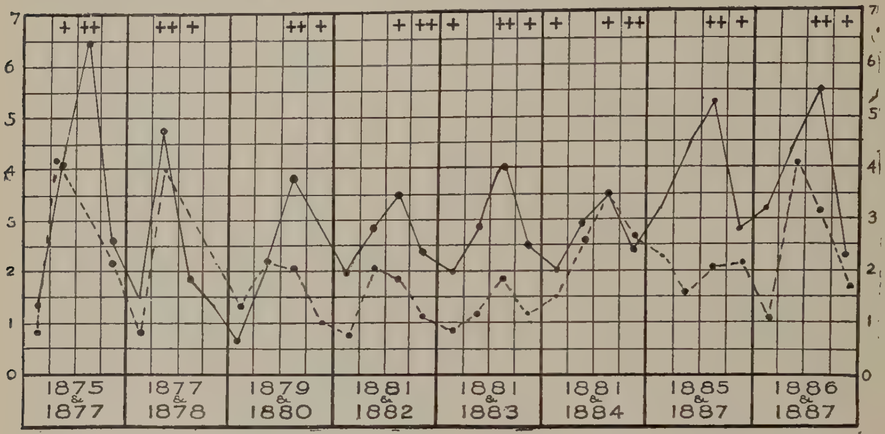

Chart No. 2.—Number of deaths of white children of five years of age and under per 1,000 of living population. Key West, Florida.
———— Yellow fever years.
.... No fever years.
+ Quarters in which cases of yellow fever occurred.
++ Quarter in which greatest number of yellow fever cases occurred.
1867: '78: '80: '81 and '87 were yellow fever years. (John Guitéras, Op. Cit.)

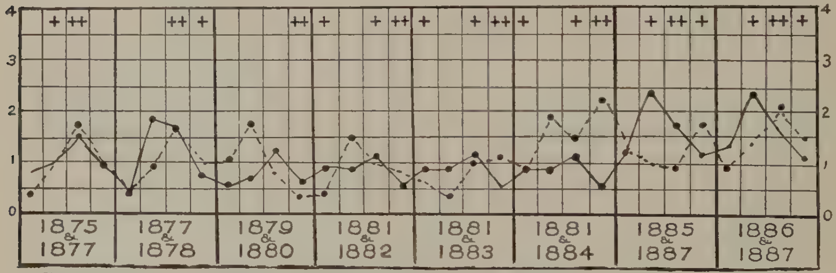

Chart No. 3.—Number of deaths of colored children of five years of age and under per 1,000 of living population. Key West, Florida.
———— Yellow fever years.
.... No fever years.
+ Quarters in which cases of yellow fever occurred.
++ Quarter in which greatest number of yellow fever cases occurred.
1875: '78: '80: '81 and '87 were yellow fever years. (John Guitéras, Op. Cit.)

The increased number of deaths ascribed to yellow fever in 1878, however, does not account for the excess of mortality, 544, for the trimester. These, it is reasonable to believe, were caused in the same way as was the excess in 1858 and 1867, in which years Chaillé gives no record of the deaths of children due to yellow fever.

These charts by Guitéras, made out for Key West, present the same phenomenon graphically. They are less striking because Key West was a small place and the intervals between epidemics of yellow fever were rarely long enough to allow a susceptible native population to accumulate. On the other hand, they do not show the disturbing influence due to the immigration of susceptible people. Both these and Chaillé's tables show the difference in mortalities of the blacks and the whites.

Yellow fever in children, then, is oy no means without fatality, and this fatality is beyond comparison greater among white than among colored children. On the basis of these two propositions Guitéras makes an "indicator" that may show the presence of unrecognized or concealed yellow fever in a community. Briefly, it consists of a tabulation of the mortuary statistics for a number of years. These are arranged in groups according to age intervals, and each group further classified into two: (1) under such diagnoses as experience has shown that unrecognized cases of yellow fever are apt to be reported and (2) under other diagnoses which cannot be readily confounded with this disease. The first includes acute fevers; acute nephropathies; acute gastrites; *embarras gastrique febrile;* diseases showing hemorrhagic symptoms; diseases showing jaundice: acute cerebral affections, etc. As far as possible the years in which epidemic diseases of childhood prevail are not included, nor those years in which yellow fever is known to have occurred. Thus a picture of the normal mortality of children in the town is obtained which on being compared with the mortality of any especial year may show (1) the increase in mortality of children, (2) the diagnoses responsible for that increase. These two factors give us valuable data, which may by themselves enable us to determine if yellow fever existed during the year in question, or at least so direct our inquiry as to determine it. If the record shows also the race, as do those of the United States, Brazil and Cuba, and if we find that the increased mortality is confined to *white* children and presents the confusion-diagnoses in the cases of these children, while at the same time the mortality of colored children remains the same, the case is clear for yellow fever. If this picture is *not* shown and there is no disturbing factor, such as an epidemic of measles or whooping-cough, the existence of yellow fever, to an extent affecting the mortality of children, may be excluded. The record of the race in mortuary reports is thus of extreme importance in countries where yellow fever may prevail. Unfortunately, in some Spanish American countries there is a strong sentiment against such record of race. "They are '*blancos*' and '*morenos*' all their lives, but after death we are all only *cristianos*." This is a kindly sentiment, but from a sanitary standpoint, unfortunate. This method of Guitéras is especially valuable in places where yellow fever is supposed to be mildly endemic, in which, as is characteristic of such places, periods of epidemicity alternate with periods of comparative freedom. In such places, in fact, its value can scarcely be over-estimated.

Immunity.—The general question of immunity to yellow fever will not be discussed here; yet the yellow fever of children has played an important rôle in determining the doctrine we now hold in regard to this aspect of the disease. In fact, the papers of Chaillé and Guitéras, from which I have drawn the above tables, were written to sustain the thesis that immunity to yellow fever is conferred in no other way than by an attack of the disease.

Immunity by Birth.—That immunity is conferred by birth in an endemic focus of yellow fever; and **Immunity by Residence**—that immunity is conferred by continuous residence for a period of years in such a focus—were, during the early part of my professional life, held without question by the physicians of New Orleans and Spanish America. "Mithridatism," as this process of acquiring immunity was called, was ascribed to the inhalation of the "poisonous atmosphere" of such places until immunity to the disease was acquired. French writers, especially Ruf z de Lavism and Lota of Martinique) less definite in their attitude to this theory, at times clearly *implied* a belief that the "immunity of the Creoles" was due to previous unrecognized attacks in childhood; positive and explicit statements to that effect, however, were lacking. Such explicit statment as *was* made asserted the doctrines generally held—*i.e.*, immunity by birth or by residence.

The monograph of Chaillé ("Acclimatization or Acquisition of Immunity from Yellow Fever") in 1880, *proved* that immunity was not conferred by birth in New Orleans by demonstrating that in epidemic years yellow fever was common, although unrecognized, among native-born children. That of Guitéras ("Some Observations on the Natural History of Epidemics of Yellow Fever Based on a Study of Mortality Statistics of Key West"), written in 1888, showed the same conditions existing in Key West and extended the argument, although not by direct evidence, to all endemic foci of yellow fever. These two papers are convincing. I know of no recent writer who claims any other mechanism for immunity to yellow fever than an attack of that disease.* This conclusion is not only universally accepted, but in places that have been investigated has, moreover, been *proven.* We can then count this matter settled. *Immunity to yellow fever is acquired only by a previous attack of the disease.*

From this we deduce the conclusion, that when we find a person immune to yellow fever (natural immunity being very rare) such person has already had the disease. If all of a community above a certain age is immune to yellow fever, every person above that age (except the few naturally immune) has had yellow fever. Thus in Bahia Brazil during an epidemic in 1912, 1913 and 1914, no case was reported in a native over 10 years of age, nor in anyone born elsewhere who had been a resident of Bahia over five years. The deduction in this instance is that yellow fever up to 1915 had been present in Bahia with sufficient constancy, or frequency, practically to insure an attack in a native within a period of 10 years from birth, or in an adult within the first five years of residence. It is fair to say however that this condition no longer exists. In Havana an examination of the hospital records for some 15 years prior to 1900 showed only one case of the disease occurring in an adult after four years residence. Most of the cases appear within the first or second year. There was no record of any case in native-born children. The interpretation of the first fact is the same as for Bahia; of the second, that yellow fever in native-born children had not been recognized.

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* Yet at the Pan-American Scientific Congress of 1915–1916 at Washington, one of the delegates—Section Medicine and Hygiene—brought in a resolution, the preamble of which asserted immunity by birth in an endemic focus of yellow fever. He was a young man, a physician of high standing, and resident in a city which was an endemic focus!

Immunity seems inextricably bound up with the yellow fever of children. We have seen how immunity is acquired. How is it lost? Or is it lost?

Whether the immunity produced by an attack of yellow fever is permanent or temporary is a disputed point among modern epidemiologists. That it is permanent has been, and is now, the opinion of American epidemiologists and also of the older generation of French and English writers, who added so much to our knowledge of this disease by their observations in the West Indies and in Africa. There was, however, a very common belief among the laity of localities in which yellow fever was endemic that leaving such focus of endemicity for a sufficient time would restore the susceptibility to the disease. This belief is alluded to by many writers. It was, I think, universally regarded as erroneous by those *not* living in endemic areas, but given more consideration, and, I think, generally affirmed by writers who lived in such areas.

It is fair to say that this latter view is the one advocated by most of the modern writers. A Commission of the Pasteur Institute working at Rio states categorically, "that yellow fever is kept up in endemic centers by recurrent attacks among the indigenes." This view is likewise accepted by Rubert Boyce: Seidelin and the British physicians in Africa.* With this opinion, however, the writer does not agree, considering the contrary proven, in so far as a negative, *i.e.*, that recurrent cases do *not* occur, can be proven. It would take too long to discuss this question and present the necessary evidence here, which is set forth in an article published in the Annals of Tropical Medicine and Parisitology, Liverpool School of Tropical Medicine of April, 1916, x, No. 1, "Immunity to Yellow Fever." This question, as will be seen later, is of prime importance not only in the epidemiology of yellow fever, but in its sanitation as well.*

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* In spite of quoting the opinion of the Pasteur Commission and giving it much weight, Clarac and Simond say: "For our part, if it be permitted to be absolutely affirmative in such a matter, we would say that a grave yellow fever, or even one of moderate gravity, never recurs." (Fièvre Jaune: page 142. Traité de Pathologie Exotique, cxi.)

Assuming then, that one attack of yellow fever gives permanent immunity, let us see what must result and how the result thus deduced agrees with observed facts.

Disappearance of Yellow Fever from Failure of the Human Host.—From the known facts of the conveyance of yellow fever, it is obvious that the conditions necessary for the continued existence of the disease in a community are three: The causative microörganisms or the parasites: Active Aëdes (Stegomyia) egypti and susceptible men: All present at the same time with access of the insect to the other two.

The parasites exist only in an infected mosquito or in an infected man. They live in the mosquito only during her life. Their existence in man, so as to be infective to mosquitoes, is comparatively short.†

Here then, are two postulates:

1. Since the parasites in the mosquito live only during the life of the host, say *X* days, no interval greater than *X* days may elapse between the date when some sick man infective to mosquitoes is bitten by them and the date when one of the mosquitoes thus infected feeds on a man susceptible to yellow fever. Without the death of the parasites and with this the extraction of yellow fever in that community.

2. Susceptible people, therefore, are necessary for the continuance of yellow fever in a community. Such people must moreover be present under certain conditions of time and place with relation to the infected Stegomyia. If in a community there be no susceptible people fulfilling these conditions yellow fever will disappear.

Now let us consider a community in the Tropics in which there is yellow fever; susceptible people and abundant Stegomyia, active at all seasons. Parasites, of course, are present in those sick of yellow fever, and, since Stegomyia are active all the year round, this place will be an *endemic focus of yellow fever.*

If, as we hold, one attack of yellow fever produces in general a permanent immunity, obviously such a community unless there be an influx of new people will have in time no one left susceptible to the disease. Then, as soon as the infected mosquitoes died off (within our X days), the parasites would disappear and the community be free from infection. Once free, it would remain free forever, unless the same three factors for conveyance are again brought together. Indeed, the disease would doubtless disappear before there were no people susceptible to yellow fever left, because, under the doctrine of chances, there would be no susceptible people left fulfilling these conditions of time and place. In the natural course of events a new generation would grow up, susceptible immigrants move in, and Stegomyia breed without limit, but unless the *parasite* be again introduced, the community would remain free from yellow fever. In such a community, developing thus naturally, an epidemic would break out if parasites were introduced some years after it had been free of infection. The maximum age of the natives then developing yellow fever would depend on the length of the previous interval of freedom.

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* By the statement that permanent immunity is conferred by one attack is meant that this is generally true, as of small pox, measles, etc. That permanent immunity is invariably conferred by one attack is not predicated of any disease. "The Absolute does not exist in the matter of immunity."

† To the writer the evidence for the existence of a reservoir-host other than man and the mosquito, seems to have no basis in observation. There is, of course, analogy for it. The evidence—of necessity negative—against it in places in which yellow fever has been studied is considerable. With the data extant the above statement seems justified—in the Americas at any rate.

An immigration of susceptible people, then is necessary for the continuation of yellow fever in a community, and if this immigration does not occur, or fails to fulfil the specified conditions, yellow fever disappears. This condition the writer has called: "The disappearance of yellow fever by the failure of the human host:" (Page 24, Supplement No. 19, Public Health Reports: "Yellow fever. Its epidemiology and control." Lectures delivered, etc.)

This immigration might be of two kinds, consisting either of susceptible people from some other place, or of infants born in the place itself, of whom, as Blair has said "truly are they new-comers" and comprise an element fully as susceptible to the disease as the regular immigrants. If these additions to the susceptible population *conjointly* fulfill the conditions necessary for the continuance of yellow fever, as I have stated them, the disease will continue; if they do not, it will disappear.

The effect upon the propagation of yellow fever of each class of these additions to the susceptible population, *i.e.*, men from outside the community and babies born within it, will depend on many factors, among others upon the amount of such immigration, increasing for each class with the growth of that class, but not directly proportionate to these increments.*

Both classes of immigration then, affect the continuance of yellow fever, and theoretically either one may be sufficient to continue it. Yet the *proportional* effect of the introduction of an adult and the birth of a baby in maintaining the infection of yellow fever is very different. The average baby for say, six months or a year, seldom leaves the premises in which it is born, and hence its chance of contracting yellow fever from *any other place* is small. If it does contract yellow fever from *that* place it will, in all probability, be kept there, and the only bad effect of the disease will be to continue for a short time the infection of a place already infected. There will thus be slight chance of the disease being spread elsewhere. An adult, on the other hand, goes into many parts of the town, and since Stegomyia feed both by day and by night, his chances of contracting yellow fever are many-fold greater than those of an infant. Furthermore, the probabilities are that he will contract the disease in one place and be sick in another, thus carrying infection to the place in which he is sick. He may also be sick some hours, even a day, before he goes to bed and thus infect Stegomyia in a number of places. Finally, if he lives among susceptible people, that is new-comers, as a stranger is apt to do, he is likely to establish a long continued focus of infection.

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* A mathematician would say that the effect of each class varies as a direct function of the number added by that class, but that the functions of the two classes were not the same and that their joint effect varies as some other functions of these numbers, but always direct functions so that both sides of the variation increased or diminished together.

On all accounts then, the effect of an adult immigrant in continuing yellow fever in a community is greater than that of a baby, so that a larger number of babies would be required to keep up yellow fever than of susceptible adults.

Besides the influence of infancy for our native-born baby, we must consider his environment. As yellow fever is permanently endemic in the community, all adults native-born or of reasonably long residence are immune to it. In fact, the only yellow fever affecting adults is to be found among the new-comers. An infant born to an immune household is, then, extremely little exposed to the disease. He is protected from it by what Guitéras characterizes as "the shield of immunity" of his environment. True enough, his brother, five or ten years old and not yet immune, may contract yellow fever elsewhere, be sick at home, infect the mosquitoes there, and thus convey the disease to the infant of the household. Yet the child's chance of escape, already good on account of infancy, is much increased by this "shield of immunity", which surrounds him. His chance of contracting it as he grows older, however, is a certainty, if he lives and yellow fever continues in the community.

Now in such towns, the great majority of infants are born in immune households. It is obvious therefore that to supply the susceptible people necessary to keep up yellow fever it would require a much larger increase of the population through birth than through adult immigration. The latter, therefore, is of supreme importance in keeping up yellow fever, far more so in proportion to its number than the native-born infants. In general both acting conjointly are factors in maintaining the existence of the disease.

Now what facts do we find consistent with the above theoretical deductions? Yellow fever *has* disappeared from a number of the towns of the tropics in which no sanitary work has been done and in which Stegomyia is still abundant or before sanitary work was done and while the mosquito was abundant. Corinto was unquestionably infected with yellow fever in 1905, for we received passengers in

Panama who had contracted the disease in that port. We had an inspector in Corinto up to 1911. No sanitary work was ever done there, and Stegomyia were abundant all the time, although no evidence of yellow fever was seen. In 1912 we landed some 5000 marines in Corinto, who made the place a base of operations. No yellow fever appeared among them. Evidently the town was not infected. Yellow fever disappeared from Bocas del Toro *before* any sanitary work was done there and while Stegomyia were abundant. In San Juan, Porto Rico, where Stegomyia are abundant, the disease disappeared before the American occupation. After the focus of Havana was removed, the same thing happened in many Cuban towns: Matanzas, Villa Clara, Puerto-Principe, which had quite an epidemic in 1899, and even Cienfuegos. Carthagena, Baranquilla and Santa Marta (which are now free of the disease [?]) were all infected not many years ago. The first, long notorious as an endemic focus, was the port through which the susceptible travel between Spain and the Colombian plateau passed, and later was an entrepôt for troops to Panama and for people from the plateau going to Europe. Stegomyia were numerous and active in Carthagena; present in fair numbers, but fewer, in Baranquilla. Yet this region—the Magdalena Delta was shown in 1916 after a careful examination, to be free from yellow fever. In fact it had been free, certainly since 1911. In 1916 no fever was found in Bucaramanga (Columbia) which registered its first severe epidemic in 1911–1912; nor in Cucuta, nor in Maricaibo, both formerly of evil fame, but the same search was not made in them as in the towns of the Magdalena Delta; so they cannot be included in the evidence. It was certainly absent at La Guaira, where Stegomyia abound. It is not fair to put Caracas in this list because antimosquito work was carried on there during the epidemic of 1911–1914, although Stegomyia were still moderately abundant in 1916. Yellow fever was epidemic in Bahia, introduced from New Orleans, in 1849 to 1851; "Over 100,000 persons being attacked"—an estimate presumably. It lasted until 1862 and then disappeared completely, to be re-introduced from Havana in 1869.* It scarcely seems necessary to mention the disappearance of yellow fever from the Windward Islands: St. Thomas, Dominica, St. Lucia, St. Vincent's, and Barbadoes; or from Georgetown and Demarara, at all of which places it had been epidemic prior to 1900. At none of these places was its disappearance due to sanitation—unless we so consider the cessation of European immigration.

This disappearance of yellow fever is certainly a not uncommon phenomenon in tropical countries. Indeed, for the disease to disappear completely from isolated communities of small or moderate size, in which there has been little susceptible immigration, where no sanitary work has been done, and in which the Stegomyia are still abundant, is not the exception, but *the rule.*

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* This disease is reported to have been introduced once before and to have disappeared, but the nature of the disease, of which this is predicated, is questioned —I do not question it: The description given in contemporary documents (da Rocha Pitta) could only apply to yellow fever.

The writer attributes this disappearance to the lack of a sufficient number of susceptible people to continue the life of the parasite, that is, to the "failure of the human host." The *fact,* that yellow fever does disappear from towns and districts in the tropics without sanitary work and with mosquitoes abundant, cannot be questioned; there are too many instances on record. The explanation given above is believed to be true also, because (1) it is logical and accords with what we know and (2) none other is apparent. Nevertheless, it is but a deduction from a fact and not the fact itself, and as such is submitted to tropical sanitarians for criticism and investigation.*

It is obvious that this explanation depends *absolutely* on the doctrine that one attack of yellow fever confers immunity against another attack. In proportion as that immunity is permanent and general, the chance of exhausting the supply of susceptible human material by a definite number of cases of fever that is at a definite time, to the point of causing the disappearance of disease, is greater than if the immunity be of short duration and uncertain. If this immunity be not permanent but yet endures for some time, it is evident that the disappearance of yellow fever by the mechanism I have outlined can still occur. It is also evident that the men who lose their immunity through lapse of time (or any other cause) keep up the supply of susceptible material just as immigrants would. In proportion as attacks recur frequently and at short intervals, the chance of "failure of the human host," to the point of causing disappearance of the disease, diminishes. If "frequently recurring attacks of the indigines" are the rule and these recurrences are indefinite, they *might* very well continue the fever in a very mild or "larval" form indefinitely; independently of immigration or new births. The *possibility* of disappearance by the mechanism we have given is then not dependent upon the permanence of the immunity given by one attack, but the *chance* of its occurring in any place at any definite time is directly dependent on it. The frequency with which yellow fever has disappeared when immigration was lacking is evidence against recurrent attacks.

It is to be noted that the localities which did not free themselves of the disease have been large towns and with a considerable amount of susceptible adult immigration—such as Guayaquil, Havana, Rio, Panama, etc. The same is true, to an extent, of places so situated in relation to towns infected with yellow fever that there is opportunity for frequent introduction of the parasites; hence the use of the word "isolated" in a previous paragraph.

The travel-relations between a number of small towns can well be such as to make the region of the combined communities a permanent focus of yellow fever, even if no one of them by itself constitutes such a focus. The conditions in these combined communities must allow the mutual interchange of infections (parasites) and yet no exhaust the number of susceptible people necessary to continue the disease, in all of the towns at any one time. In such an area the rapidity of spread of the fever, and hence the exhaustion of the susceptible material, would be much slower than if all the people were in one town. There might in such a community be intervals between infections in any one community long enough to allow a considerable number of natives susceptible to yellow fever to accumulate. This would give rise to a series of small epidemics among the children; the age at which they would be attacked depending on the length of the interval of freedom from yellow fever. This may explain the persistence of yellow fever in Yucatan and Campeche, and in Africa. This condition appears to have existed in the litoral of the Caribbean Sea before Havana became a permanent focus in 1761. At that time, the area *as a whole* was a permanent endemic focus of yellow fever. One part after another would become infected, but no single one of them would suffer continuously. Such a conception seems to the writer the best explanation of the history of yellow fever on the Spanish Main and in the West Indies during this period. There is nothing unusual in this conception. It is analogous to the continuous existence of measles in a section of country comprising a number of towns, no one of which is always infected with it. The elimination of the insect host in several, or even one, of these communities, to such a degree as to make them not infectable with yellow fever, may so break the chain of transfers of infection, as to allow all the rest of this communicating area by the failure of the human host to free itself permanently. Anti-mosquito work then at even a few of such places may give good results out of proportion to its extent.

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* It is at present 1922 universally accepted.

The effect of a permanent focus, such as Havana became in 1761, was not to *prevent* other communities from freeing themselves from yellow fever by the mechanism we have outlined, but to *re-infect* them as soon as a sufficient number of susceptible people, by birth or immigration, had accumulated to allow of propagation of the fever. These smaller places then, show a series of small epidemics with intervening periods of freedom. The towns on the Guayaquil and Quito Railroad and on the West Coast of South America in communication with Guayaquil, illustrate this phenomenon beautifully. There was an epidemic in Buenaventura in 1903 and 1904, followed by several years of freedom. Then in 1914 epidemics occurred in Tumaco and Esmaraldas, followed in 1915–1916 by another, in Buenaventura when only "children and people who had moved in during the last eight or ten years" were attacked.

It is this recurrence of fever which has given rise to the belief, held by many very eminent men in "larval" yellow fever. Unquestionably, yellow fever may, and at times does, exist unrecognized among the native children of a community, being recognized only, when it attacks some stranger. Here we have true recrudescence whenever an influx of strangers occurs. This view is undoubtedly true and is too well known to require elaboration. The writer will not pretend to predicate how long such a condition may continue, nor deny that, under some conditions, it may last indefinitely, thus maintaining the place as a permanent focus of yellow fever. What he does deny is that this condition is the rule. The instances given prove that the spontaneous disappearance of yellow fever is not rare. An analysis of them, if there were space, would show that in the absence of adult immigration and of inter-travel (if one may coin a word) among them, the disappearance of yellow fever is to be expected to occur in a large proportion of towns, and that then, even when there are influxes of strangers, outbreaks in such towns do not occur unless they are in communication with some infected focus. These outbreaks, then, are re-infections and not recrudescences of larval yellow fever.

In order, therefore, for a town which has freed itself from yellow fever by the failure of the human host to remain *permanently* free, *isolation* from infected places is necessary. When yellow fever has been eliminated by *the control of the insect host*, this isolation is not necessary *as long as this control continues to be efficient*, because, in the absence of the insect host, yellow fever is not communicable, and such parasites as are brought in by infected men or infected mosquitoes would, at the most, establish a very temporary focus of infection. If the control were complete, infected men would transmit no parasites. Men would indeed acquire the disease from infected mosquoties, if the parasites were thus introduced but from these cases there would be no transmission to other mosquitoes, and hence no secondary human infections and, with the death of the mosquitoes introduced, the conveyance of the disease would be over. It is to be noted, however, that the reduction of Stegomyia sufficient to eliminate yellow fever from a town in the tropics would nearly always be less than would be required some years later to prevent its spread when reintroduced, because there would then be a larger proportion of susceptible people than at first. Hence the mosquito control must be more intensive.*

What has been said explains the great diminution of yellow fever which is now apparent in the Americas:

1. After the extinction of the great permanent foci at Havana, Vera Cruz, Panama, and Rio, sanitary work also effected the disappearance of the disease from a number of less important places: Para, Manaos, Iquitos, probably Pernambuco, and possibly Bahia and Caracas. Such ports, especially those on the Caribbean, as could spontaneously free themselves of yellow fever, were thus enabled to remain free of the disease being no longer exposed to infection.

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* It is worth contrasting these two methods for the elimination of yellow fever. The method of election by human interference, i.e., sanitary work, consists in the control of the insect host. While the method by which it is eliminated in nature is by control (failure) of the human host. (Supplement No. 19, U. S. Public Health Reports, pp. 10 to 13.)

2. The substitution of steam for sailing vessels by enormously lessening the number of infections (parasites) carried between ports, decreased the chances of reinfecting such ports as had already cleared themselves of yellow fever. Sailing vessels frequently carried infected Stegomyia as well as infected men, whereas iron steam vessels very rarely carried the former. Hence, in spite of the shortened voyage, they were much less efficient in the transport of parasites.*

3. The European war both restricted foreign immigration and, by the commercial depression it caused, greatly lessened the movement of all people between different towns and from the plateaus to the Coast towns—thus decreasing the number of susceptible people in infected places and, by lessening travel, limiting the chance of infection spreading to places which had already cleared themselves of yellow fever. Where the parasite is absent the fever will not recrudesce even when there is a return of prosperity and an influx of susceptible people. Such people have returned to Maracaibo and to some other towns with no resultant outbreak of fever.

It is facts like the above which make us so hopeful that a reasonably well-organized effort to wipe out yellow fever will result in its complete elimination from the earth, so that it never will return. In many places where yellow fever now exists there is needed but a minimal amount of sanitary work to turn the scale against the disease and eliminate it, thus starting an endless chain for good, since, the freeing of one place from yellow fever so frequently prevents the infection of another. It is upon these facts that the recommendations of the Yellow Fever Commission of the International Health Commission have been based. The purpose of these recommendations is this complete, permanent elimination of yellow fever from the globe. This fate has befallen some of the higher forms of animal life, even in historic times, but this will be the first time in which an attempt for this purpose has been made against a microörganism pathogenic to man. The accomplishment of such an end will (I do not say "would") mark an epoch in sanitation.

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CHAPTER CXXXIV

MALARIA

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Anyone writing a treatise upon malaria is confronted with an embarrassment of riches, for the literature of the subject is enormous. The authorities mentioned in the bibliographic record have been freely consulted, but much of the matter is from mental register of knowledge acquired from special reading and much practice and teaching extending over many years. It is impossible now to catalogue the sources of this material, and this indefinite credit, therefore, is all we are able to give.

Since the outbreak of the War, our chief sources of information have become barren, owing to enormous activities in other fields. This partly accounts for paucity of very recent references ("Addenda," p. 776).

Synonyms.—Malarial fever; intermittent fever; remittent fever; chills and fever; fever and ague; paludism; marsh fever; swamp fever; tropical fever.

Some of these terms are applicable only to certain clinical types, which will be considered in detail.

Definition.—A morbid condition produced by the infection of the red blood corpuscles by one or more broods of one or more species of a hematozoon. It is characterized clinically by fever, intermittent or remittent, associated with swelling of the spleen, resulting in anemia and the deposit of pigment in the viscera, followd by cachexia with great splenomegaly in neglected cases.

The infection is transmitted by the bite of a mosquito of the Anopheline family, which in the Southern United States is usually the A. quadrimaculata.

Historical.—"Disease probably described as tertian and quartan fevers 1000 B.C. and in the Iliad and the Wasp of Aristophanes. Probably introduced into Greece from Egypt. Inscriptions on the Temple of Denderah (AAT) are supposed to refer to miasmatic fevers." Hippocrates, 525 B.C., writes: "Omitting those (fevers) arising without evident pain, there are four types, the names of which are: Continued fever, quotidian, tertian, quartan, . . . of autumn, most of the summer, quartan and irregular fevers, enlarged spleen, dropsy, etc." He says further ". . . such waters as are marshy, stagnant and belong to the lakes are hot in summer, thick and have a strong smell. Those who drink from them have large and obstructed spleens." The Romans had a temple dedicated to Mefitis, the goddess of fever and vapor. She was represented as an emaciated, half nude female, horrid, bald-headed, with a huge belly and swollen veins. In one of Horace's letters to Maecenas he begs him "to remain away until colder weather, for when the figs begin to ripen and faces become pallid with fever, the chief of the funeral pomps, with his black assistants, is very active, and the reading of wills becomes the order of the day." Thus far the history deals with the hydric theory of transmission.

The military engineers of the early Romans begin to question this. Thus Varro, 118–29 B.C., expressed the opinion that the fevers were produced by animalcules so minute as to be imperceptible to the naked eye and which enter the body from the air through the nose and mouth. The name "malaria" was given to the disease in 1753 by Torti, who on the ground of therapeutic differentiation of fevers into those that yielded to "bark" and those which were uninfluenced by it, has produced a classic that will endure for all ages. Morton and Torti had previously (in the seventeenth century) alluded to this differentiation. Lancisi, 1717, resurrected the theory of animalcular origin of Varro, and added his conviction that these minute organisms were introduced by stinging insects, which imbibed them from marshy waters. Rasori, a century later, is quoted as saying that he was convinced that the sequence of symptoms of intermittent fevers was due to the progressive development of a parasite in the body, and the varying periodicity was due to variety of species. Coming back to the seventeenth century, the period of the introduction of the bark of a Peruvian tree; in 1638 the Countess of Chinchon, wife of the Viceroy of Peru, was cured of a severe intermittent fever, and two years later her physician, Juan del Vego, imported the bark into Europe. There is no more striking example of the retardation of research into etiology on account of the possession of an efficient remedy than there is in the history of malarial fever. Meckel, in 1847, was the first to describe the pigment in the leukocytes and in the liver and spleen. Virchow, later, found pigmented bodies in the red cells. In 1849 J. K. Mitchell, of Philadelphia, created a furore with his announcement of the presence of spores in damp and marshy places and their relation to "malaria." Salisbury, in 1869, connected the spores of "palmella" with etiology. In 1879 Tomassi-Crudeli and Klebs announced that they found rather uniformly, in marshes, a bacillus which produced symptoms similar to those of malaria in experimental animals. In November, 1880, Alphonse Chas. Louis Laveran, a French Army Surgeon, announced from Constantine, Algiers, the discovery of a blood parasite and its relation to paludal fever. Marchiafava and Celli endeavored to dispute Laveran's priority notwithstanding the fact that they, as late as 1884, still maintained that the pigmented bodies in the corpuscles were degeneration phenomena. The history of malaria may be said to have been completed in 1900 when the work of Ross, Koch, McCallum, Grassi, Bignami and others definitely traced the life cycle (exogenous) of the parasite through the mosquito, and that of Sambon and Low who after spending the summer and fall under screen in the Roman Campagna in perfect health, sent experimentally infected mosquitos to England where malaria is not endemic and produced typical paroxysms in the son of Sir Patrick Manson. Schaudinn, in 1902, was able to trace the entry of the sporozoits into the red corpuscles of his own blood under a cover glass. In 1901 he announced his discovery of the parthenogenetic cycle. Craig, in 1910, described another cycle, the "conjugation phase," which he holds responsible for latent and relapsing malaria. Bass and Johns in October, 1912, announced their success in cultivating plasmodia from the blood in 29 cases of estivo-autumnal malaria and six cases of tertian, in Panama. For details of the method of cultivating malaria we must refer to the original articles of Bass, David Thomson and others.

Etiology.—It has been established that "malaria" is not bad air but an infection of the blood by parasites, introduced by certain mosquitos. The question is often asked: Which had it first? We can only assume that the extraparasitic phase of the parasite has ceased to exist.

Zoölogy of the Parasite of Malaria.—*Division.*—Protozoa: Unicellular; organisms not differentiated into tissues, thus differing from metazoa.

Class.—Sporozoa: Having the property of sporulation and two modes of reproduction, one within the host, the other for dissemination outside the host.

Subclass.—Telesporidia: Reproduction follows when sporulation is complete.

Order.—Coccidiomorpha: Intracellular; reproduction anisogamous.

Suborder.—Hemosporidia: Are within corpuscles; ameboid when within cell, spores naked.

Genus.—Plasmodium (Marchiafava and Celli), not a true plasmodium but name has priority.

Species (1) *P. malariæ* (Marchiafava and Celli, Golgi), parasite of quartan malaria, segments every 72 hours; species name has priority, being the first one named; gametes spherical when encysted; 8 to 12 segments.

(2) *P. vivax* (Grassi and Feletti), parasite of benign tertian malaria, segments every 48 hours; gametes spherical when encysted; 14 to 22 segments.

(3) *P. falciparum* (Welch, Blanchard), parasite of estivo-autumnal fever; segments usually in 48 hours; sometimes seems to segment precociously (formerly described as a species—P. praecox; presents certain morphologic peculiarities due to rapidity of development). Parasite of malignant malaria and irregular symptomatology; in compact groups gives rise to subtertian paroxysms; gametes crescentic

when encysted; often seen with bridle of red-cell substance attached, hence name. Six to 32 segments.

P. tenue has been proposed but not accepted.

Many views held as to the nature of malaria and the parasite responsible for it will be better understood if we analyze the development of our knowledge of the parasite. Laveran, in 1880, while searching for the cause of the melanemia, observed some bodies concerning which he made a preliminary report in the same year. He described them as hyaline, spherical, pigmented, non-nucleated bodies some of which, in 15 to 20 minutes, extruded pseudopodia which subsequently detached themselves "*que chaque flagellum vit à ce moment d'un vie indepéndante.*" Also bodies which were motionless or nearly so, exhibiting a collection of rounded pigment granules near the center. Next, certain forms which he considered cadaveric. In 1881 he also reported upon smaller bodies which were one-sixth the size of the red blood corpuscle and either free or attached to red blood corpuscles. Marchiafava and Celli, even in 1884, did not consider the large bodies (now recognized as gametes) as anything but degenerated blood corpuscles. They endeavored instead to establish small "micrococcus-like" bodies, within red cells, taking the methylene blue stain, which they subsequently named "plasmodia." Laveran had already (in 1881) described these as "*formes primitive, embryonnaire*" and located them upon the red blood corpuscles (*accolés*). In 1882 he emphasized "having said long ago" that the crescents and ovoids were seen most in relapses and in malarial cachexia.

In Pavia on the River Po in the North of Italy, Golgi, in 1885, added an important link to the chain. He had the good fortune to see many quartans, in which he was able to follow the various stages up to segmentation (this parasite is seen in the peripheral blood throughout its cycle) and established the relation of phases to the sequence of symptoms. The chill always came after segmentation. He considered the paroxysm as due to the invasion of new cells by the new generation of parasites. Antolisei disproved this by showing that if quinin is given before the expected paroxysm, the chill nevertheless appears but the invasion of new cells is prevented. Golgi, in 1886, made similar observations of sequence of development in benign tertian. In his locality he had not yet encountered crescents. Soon after, Roman investigators divided malarias into intermittents, caused by the "plasmodia" of tertian and quartan fever, and irregular forms, appearing in summer and fall, hence estivo-autumnal fevers. These were associated with the formation of crescents. Many attempts have been made to subdivide the parasites of estivo-autumnal fever into different species. Marchiafava and Bignami were the first to describe differences of size, character and time of development in estivo-autumnal plasmodiai dividing them into quotidian and tertian types. Grassi and Felett, made a further division describing a pigmented and unpigmented, small parasite (Hemameba precox, hemameba immaculata) and a larger one, the cause of irregular fevers (*Laverania malariæ*). Mannaberg, and very much later, Craig, supported this view, the latter describing differently shaped crescents for the quotidian variety.

Mannaberg attempted to establish the estivo-autumnal parasite as a separate genus on account of the occasional appearance of zyzygia, which he never encountered in the tertian and quartan types.

Almost all observers are now agreed, upon sufficient evidence, that no species difference exists which justifies the designation "*precox*" or "*immaculata.*" Precocious segmentation is seen, at least segmentation before full growth, giving often only five merozoites, but in cultures this has never been observed.

All authorities now agree also that gametes require more than three or four days to reach maturity. The rôle of crescents in chronic and relapsing malaria has been the subject of much study. Bastianelli and Bignami believed that they were sterile. The observation that they were associated with chronic and relapsing malaria was made by Laveran and supported by Councilman, Dock, Osler and others. The first developmental basis was furnished by Canalis, who described segmenting gametes at length. Grassi and Feletti, and Mannaberg, later, Ziemann. and Maurer observed segmenting gametes. Golgi accepted this view (third mode of division) and thinks this is seen when paroxysms occur at longer intervals (*febbri malarichi a lunghi intervalli*). In 1901 Schaudinn came forward with a description of a definite sequence of events which he described as the segmentation of macrogametes, and to which he gave the name of parthenogenesis. His view that relapses are due to parthenogenesis is still upheld by some writers.

Craig ascribes relapsing malaria to plastogamy. He believes to be justified in considering the zyzygia described by Mannaberg and Ewing as an early stage of these encysted bodies. Plastogamic figures have been seen by James in Ancon and Henson in Florida. Schaudinn says: "*Plastogamie kommt bei den Malaria-Parasiten nicht vor.*" Calkins (op. cit.) says: "It is difficult to believe that two widely different processes of fertilization should exist for the same species."

Schaudinn's figures have been seen by very few observers. The writer has seen some of James' slides. Only once in our clinic did we see such formation. I now think it might be interpreted as a blighted gamete in the same corpuscle with a segmenting body. Curiously enough, Antolisei in 1890, describing in detail what he considers Golgi's "third form" and which he regards as a degenerative process, gave a very good description of Schaudinn's segmentation, if allowance is made for unstained morphology. Deaderick accepts parthenogenesis without comment. Bass has seen such pictures in his cultures but with no regularity. Ross, the Thomsons, Simpson, Craig and others with large experience have seen no figures justifying Schaudinn's assumption. Celli says larvate, visceral sporulation can account for all the symptoms of chronic and relapsing malaria. Giemsa and Werner have never observed parthenogenetic forms in their cultures.

Cycles of Development of the Malaria Parasite.—No matter which species is injected by the infected mosquito the *sporozoits*, which are the end-products of the development within the mosquito, enter the red blood corpuscles, stripped of much of their cytoplasm, which remains in the plasma, and begin to grow. They are now known as *schizonts* (def.: Bodies capable of splitting or dividing) also: *Endogenous, asexual,* trophocyte, Monont, *nutrient body,* ondenterospore. These grow and, according to species or type, reach the stage of segmentation (also, sporulation) when the containing red cell ruptures and releases the resulting *merozoites.* These, 6 to 34 in number, are expelled into the plasma, resulting in the chill; as many as are not destroyed by phagocytes enter new red cells and complete the same cycle as many times as circumstances permit. The *period of incubation* is the length of time required to multiply their number sufficiently to produce a definite paroxysm, usually preceded by some days of malaise. After a few definite paroxysms, a portion of the merozoits within corpuscles develop much more slowly and can be seen to be much more compact when still very small. This marks the beginning of the *exogenous cycle.*

Almost from the very beginning, when they are the size of a red cell, the two forms can be distinguished. One stains darker and has a more compact nucleus; the other is looser in structure, both in cytoplasm and nucleus. The former is the *female gamete* or *macrogamete,* the latter is the mother cell of the *male gamete* which is very small, several being in each body. Hence its name, *microgametocyte,* containing the *microgametes,* or *male sexual elements.* Each gametocyte, i.e., the macrogamete and microgametocyte, grows to complete maturity within its respective containing red blood corpuscle and then remains dormant for several weeks in the circulation or in the spleen or bone marrow. In this form it is probably entirely uninfluenced by quinin except possibly after prolonged intensive use. These are the bodies which the biting mosquito obtains from the patient and which complete their cycle within this *definitive* host. *Part of the further development can be observed in fresh blood under a cover glass.* If the blood is first kept in a moist chamber for some time and then examined, the microgametocytes will be seen to become spherical and then undergo great internal commotion. Soon a process forms on the surface, becomes longer and finally escapes and lashes violently about, scattering the neighboring corpuscles; then others, and still others escape until finally the microgametocyte is a dead, vacuolated, motionless, shrunken mass. These loose bodies perform the function of spermatozoa, and are known as spermoids, *flagella* androspores, microspores, polymitus, sperm-body, more properly, *microgametes.* Within the middle intestine of the mosquito we can follow the cycle further. The microgamete enters a macrogamete, and the cell thus

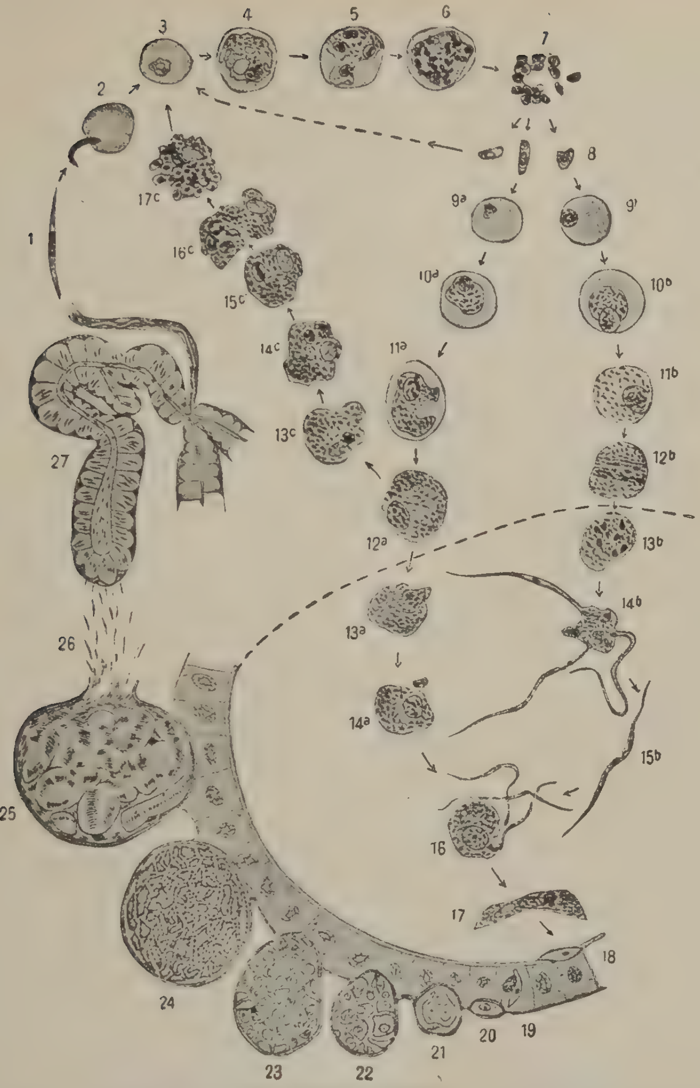


Fig. 360.—Diagram to illustrate the life-history of the human tertian parasite, *Plasmodium vivax.* (After Lühe, from Braun.) Magnification: 1–17, 1,200:1. 18–27, 600:1. 1, Sporozoite. 2, Penetration of the sporozoite into a red blood cell. 3–4, Growth of the shizont. 5–6, Nuclear division within the schizont. 7, Breaking up of the schizont into merozoites. 8, Single merozoites, one of which (left arrow) enters a blood corpuscle and develops into a schizont (3–7). After a certain period of infection the sex-forms develop: 9*a*–12*a*, Macrogametocytes. 9*b*–12*b*, Microgametocytes. If the macrogametocytes remain in the blood-stream of man, they multiply by parthenogenesis and form schizonts (13*c*–17*c*). Below the dotted line the stages which take place in the gut of *Anopheles* are shown: 13*a*–14*a*, Maturation of the macrogametes. 13*b*–14*b*, Formation of the microgametes. 15*b*, A microgamete. 16, Copulation. 17, Motile copula or "oökinet." 18, Oökinet penetrating the intestinal wall of the mosquito. 19, Oökinet passing through the epithelium. 20–25, Sporogony upon the outer surface of the intestinal wall. 26, Passage of the sporozoites to the salivary gland. 27, Salivary gland of mosquito containing sporozoites.

fertilized assumes a fusiform shape and becomes actively motile, the *zygote* or *traveling vermicule* (oökinete, amphiont, copula). Properly, it is the zygote only before it has become the vermicule, and before the nuclei have fused. In traveling it perforates the walls of the intestine into the muscle fibers of the wall where a double contour is now evident, it is encysted and known as the oöcyst. The sporoblastic stage next begins, and in subsequent stages spiculated masses are made out, from the edges of which the sporozoits are seen sprouting forth. The cysts grow to enormous size and finally rupture, discharging as many as 10,000 sporozoits. Some of these travel to the large salivary glands which fill the thorax of the mosquito, from which they are discharged through the salivary gutter of the mouth organ into the vein of the victim.

Additional Terms.—The *endogenous cycle* (schizongony, fever cycle, human cycle, asexual cycle, monogonous cycle) is the "short" cycle and occurs only in the blood and causes the fever.

The *exogenous cycle* (sporogony, afebrile cycle, mosquito cycle, amphigony) begins in the blood and continues to the maturity of the sexual differentiated bodies, gametes, (i.e., the macrogametes and microgametocytes, formerly known as "resting bodies," crescents, ovoids, spheroids) and is completed in the mosquito. The gametes of the plasmodium falciparum are crescentic and oval.

The detailed morphology of the parasites and their differentiation is given under "Examination of the Blood."

The Malarial Mosquito.—(The family of anophelines has been divided into several subfamilies.) In the United States we are concerned mainly with three species: *A. pseudo punctipennis; A. quadrimaculata*, and *A. crucians.* All three can be differentiated by their wing markings.

The males have bulbous palpi; the palpi of the female are as long as the proboscis. The antennæ of the males are plumed. Culices rest on the wall in a "humped" position; anophelines are straight, slender, and rest in the "brad-awl" position.

The anopheline ova are single, with lateral floats, and so small as to be practically invisible without magnification. The eggs of other species are in rafts and the individuals vertical to the surface. The anopheline eggs are laid on the surface of the water and in floating about assume fanciful patterns. The egg stage in the most favorable weather continues 48 hours, while in unfavorable weather it may remain more than twice as long.

The larvæ remain horizontal, just as they are hatched. The larva must be differentiated from that of other species wherever intensive anti-malarial campaigns are made. The larva lies on the surface, horizontally, is light in color, except for its black palmate hairs which serve as fins. It has no siphon.

Breeding Places.—Anophelines prefer clear water with a "natural bottom" near habitations. Pools containing bordergrasses or rushes afford protection from tadpoles and minnows, and from oil. When driven to it, they may breed in water barrels and fire buckets, but they avoid stagnant pools, tin cans, roof gutters, bottles, etc., (a popular misbelief). In small streams having areas of zero current, in "potholes" of drying creeks and in "borrow pits" along railroads, larvæ may or may not be found. Ducks and top minnows feed upon larvæ. Concrete sides to necessary bodies of water prevent oviposition.

The larval stage in the southern United States lasts longer than two weeks, and becomes a practical point in antilarval work.

Contributing Causes.—Climate. Geographic Distribution.—Many sources of information mention countries from which malaria has been reported but which has been contracted elsewhere.

The morbidity census for the United States is unreliable. Tables in P. H. Reports for 1916 show reports from 11 states only, not including eight southern states. In one state the sickness and mortality returns made a death rate of 36 per cent. Figures prepared by v. Ezdorf show high endemicity in the drainage area of the lower Mississippi and its tributaries, and in all flat areas of the southern states. Small foci exist in southern California and in the Atlantic coast states as far north as Delaware, with occasional outbreaks north of Long Island. In the central states, territory north of the Ohio River furnishes a negligible amount of malaria. Any climate in which anopheline mosquitos can breed is potentially malarious. The degree of prevalence in other countries is a matter for reference to special publications.

Plasmodium falciparum has a more restricted distribution than *P. vivax*. It is the parasite of "tropical malaria." *P. malariæ* is confined to small endemic foci. Human blood is resistant to the parasite and gametes are not readily formed. A table in Deaderick and Thompson gives the relative distribution of the three species.

The influence of climate upon the parasite within the human host is interesting. Persons heavily infected often recover spontaneously after a short sojourn in the mountains. Larvate malaria will come to the surface and frank paroxysms terminate the infection. Exposure to the sun often brings on a chill. Exposure to cold, getting wet and other hardships precipitate a chill.

Soil.—Ground water predisposes to mosquito breeding. Outcroppings at hillsides may accumulate lower down and inundate grass and weeds, making the best possible breeding places. Turning up the soil has for ages been regarded as the cause of outbreaks of malaria. Groups of laborers sleep in tents and bunk-houses and the gamete carriers among them soon infect mosquitos which in time inoculate the whole gang. "Borrow" pits, potholes, small ponds and eddies in small streams are ideal breeding places.

Rainfall.—On the Panama Canal rainfalls made anti-mosquito work very difficult. High water carries the larvæ farther downstream, a surprising number remaining on the way, held by vegetation, which finally develop in small potholes, etc. Rain is fatal to mosquitos in flight if they fail to find shelter. Rainy periods usually precede the period of highest prevalence.

Season.—The seasonal variations of malaria show in a striking manner the dependence of transmission on mosquitos. The first vernal malarias are relapses. Mosquitos do not hibernate in the full meaning of the word. They come out at intervals when the weather is warm and feed. Mitzmain has shown that plasmodia do not develop within the mosquito at temperatures below 55°F. He found that although gametes were found in central Mississippi all winter, no efficient development within mosquitos could be found. Moreover, these early malarias are never *falcipara*, which is the last species of the winter. These do not relapse until later in the season. *Falcipara* may be found in Tennessee as late as February. In March and April on very warm days cases of chills are observed all of which are *vivax*. Mosquitos feeding on these furnish our first transmitted malaria. Falcipara require hotter weather to leave the bone marrow and, even then, the gametes do not thrive in mosquitos until July, in our climate. Every season is "open season" for *P. malariæ*. Ruge says one of the maledictions of the old Romans was, "quartana te teneat."

The following table from the Memphis General Hospital shows the seasonal prevalence. On account of inability of interns to distinguish species in all cases, no attempt was made to classify them:

January	5 cases	May	12 cases	Sept	139 cases
February	3 cases	June	29 cases	Oct	24 cases
March	2 cases	July	58 cases	Nov	49 cases
April	6 cases	August	143 cases	Dec	6 cases

Most of these cases originated in the contiguous uncontrolled area.

The Laboratory for Field Investigations of Malaria of the U. S. P. H. S. in one year examined 2032 diagnostic slides. Of these, 447 were positive. The accompanying chart gives the curve of incidence:

Just why malaria contracted in the spring should remain dormant until the following spring, while during the intervening period another species should dominate the field is still a biological paradox. One cannot escape the conviction that curability of malaria is only relative and that parasites are rather likely to resist both treatment and normal defenses for many months. The theory of mutability of species still finds adherents.

Winds.—New foci of malaria are started by the human host, never by the mosquito. If malaria is to be eradicated, treat the breeding places where people work, where they sleep and where they visit rather regularly. Mosquitos do not leave their home breeding places unless they are mechanically carried in vehicles and sleeping cars. Le Prince marked malaria-bearing mosquitoes and never found them far from the point of release. Wind-borne mosquitos are not transmitters of malaria.

Race.—Statistics from the War Department show that the percentage of clinical malaria among whites is three times that among blacks. It is generally held that anophelines do not bite negroes as eagerly as they do Caucasians, and it is also common observation that they attack newcomers more freely than old residents.

Age.—Cardamatis says: "Morbidity statistics of infantile malaria reveal a difference of 25 to 40 per cent. between clinical and microscopic diagnosis, which is certain proof that other diseases are often erroneously diagnosed as malaria. Experience based on microscopic evidence proves:

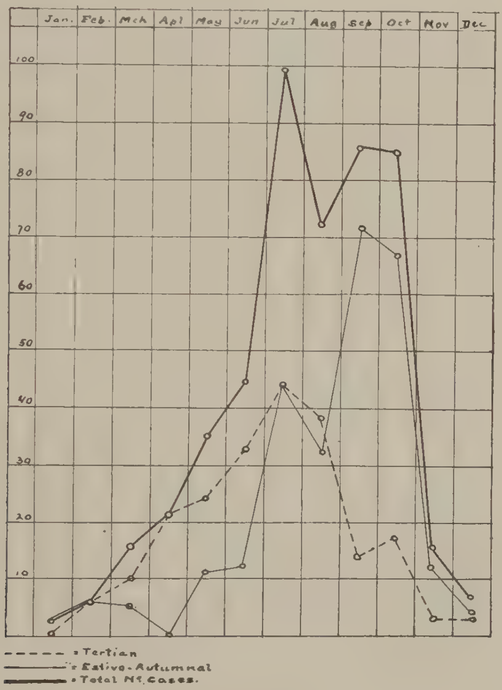


FIG. 361.

1. "During nursing period, children are less prone to attack than at later stage.

2. "During the first three months of life infantile malaria is less frequent than in the following months of the first year.

3 "A relative immunity exists during the first month.

4 "The morbidity rate is lower in the first year than in later years, probably because during this period the infant is carefully protected from mosquito bites during the greater part of the day.

5. "Malaria attacks almost equally in the early period as in later periods of childhood, particularly during the second year of life."

Congenital Malaria.—On account of the conflicting data in the textbooks it has been deemed advisable to search the literature exhaustively.

Bass (M. H.) finds consensus of opinion that malaria is probably not transmitted in utero, strengthened by negative results in search for parasites in fetal blood, cord, or fetal surface of the placenta by Thayer, Pezopoulos and Cardamatis, Marchiafava and Bignami. A small number of cases have lately offered incontestable proof that ante-natal transmission is possible although rare (Addendum, p. 776).

Meara and Beekel reviewed the literature fully. The latter collected 11 cases as follows: Duchek; Crandall (N. Y. Polyclinic, 1893) in 18 hour infant, plasmodia found third day; Hitté (Montpelier thèse, 1902), two cases, plasmodia found in blood of cord; Economos (Bull. Soc. d'obst. (1907), p. 70) 7 cases, in 5 plasmodia in fetal blood at 8 to 48 hours after birth. Dumollard and Viallet (Bull. et Mém. Soc. Méd. d. Hôp. Paris (1909), p. 229) reported a case of precipitate labor at seven months in woman undergoing treatment for malaria. Infant lived one hour, plasmodium falciparum in blood of mother, of cord, placenta and fetal heart. No hemorrhage at labor.

Lafont (Paris thèse, 1910) adds an undoubted new case.

Tissier and Brumpt (Arch. Men. d'obst. et Gynécol., iii (1913) p. 166) cite in addition the case of Dumollard, Lemaire and Lafont, of quartan fever with demonstration of plasmodium malariæ and report one case of tertain malaria (plasmodium vivax) found in blood of mother and infant. Infant developed first symptoms on nineteenth day but infection after birth believed to be out of the question as mosquitos and malaria are unknown at Neuilly in October.

Bass adds personal case: Mother 26 when about five months pregnant, had fever on alternate days. After a severe chill, was under medical care for a *few days*, with no later attacks. Infant born December 23, New York City, no hemorrhage. Infant well for month, then fretful, numerous green stools, occasional vomiting, loss in weight. Hemoglobin 10 to 20 per cent.; P. vivax in great numbers. Lymph-nodes enlarged, liver and spleen enlarged.

Cites another case: Woman who some time before had an attack of malaria was seized with chills and fever before birth of apparently healthy child. Plasmodia in mother's blood and in infant's immediately after birth. Prompt response to treatment.

Bass sees no reason why his case should not be considered a true case of congenitally acquired malaria, as post-natal infection is positively excluded. How the parasite passes from mother to fetus is unknown since careful work fails to show instance of passage through intact placenta. Small areas of placental separation may account for the infection of the fetus.

On the other hand, if intra-uterine infection had occurred, the child would have shown cachexia or anemia. All cases reported to date show *healthy infants in the first few days.* This would favor a theory of infection during labor rather than pregnancy. As to the manner of infection there seems to be no definite answer possible.

Weston says: "In endemic districts, abortion or premature labor is the expected result of malarial paroxysms. Full-term infants of malaria-infected mothers are under normal weight and height, generally weak, with poor resistance, often succumb to inanition. (Thornhill: In Ceylon in 1896, 40 to 50 per cent. of deaths under one year were due to convulsions, mostly malarial. Laveran, quoting LeBrun, says, of 2073 cases at Beirut, 1065 were children under eight years.)

"Malaria may be latent in infants for a long period without any symptoms, except anemia. No race immunity nor sex insusceptibility. Congenital malaria is still a mooted question. Convulsions frequent, almost continuous, at autopsy great numbers of parasites found in brain which accounts for this. Never heard of hemoglobinuric infant."

Thayer (op. cit., p. 96) reports the case of a colored woman with triple quartan who gave birth during a paroxysm to an eight months child. The infection in the mother's case had lasted at least five months. While the blood of the parent showed three groups of parasites, the child's blood upon repeated examination was quite free from parasites or pigment. Examination of the placenta showed pigment and parasites upon the maternal side only.

Clark (H. C.) Jour. Exp. Med., xxii (1915), p. 432, says: "Many of the cases now reported in the literature as congenital malaria suggest immediate post-natal infection as their history, as our own pathological and clinical records justify."

The following is from Cardamatis:

Congenital Malaria.—Almost all authors of ancient and modern times admit that malaria is transmitted from mother to fetus by way of the placenta. Hippocrates, Schuring, Hoffman, Stokes, Bouchut, Trousseau, Laveran, Jules Simon, Economos, are cited among others. Scheube, Freytag, Ven der Elst deny the possibility but report that infants born of malaria infected mothers have malarial cachexia and splenomegaly.

The microscopic investigations of Guarnieri, Bastianelli, Marchiafava, Bignami, Sereni, Tornu, Caccini and Thayer contribute largely to proof against transmission of the parasite from mother to fetus by way of the placenta. Only theoretical reasons are given for upholding the opinion, some based on clinical experience, others on laboratory investigations in anthrax, pneumonia, typhoid, and eruptive fevers. Clinical experience has shown the transmissibility of variola and syphilis, confirmed by laboratory research, but in all these cases transmission was due to alterations in the placenta. To pass the placenta (considered a veritable filter by Brauell, Davaine and Charrin) it is necessary that the microörganisms find the placental defect, and that they produce disturbance of the placental circulation.

In syphilis, as in other infectious diseases, abortion due to alteration of placenta is frequent.

Pascuali and Bompiani in 1884 were the first to note alteration of the placenta in malaria. Later Louros and Petsalis confirmed this, and Louros attributed the abortion or premature labor essentially to the anatomic change, i.e., extravasation of the blood, hemorrhagic and white infarct. Petsalis thinks it does not follow that histologic changes are present in the placenta of all pregnant women infected with malaria; of 18 examined, 11 showed white infarcts, varying in number from 1 to 10. But both of these last authors failed to examine the blood to prove that the mother was infected. In the only case examined microscopically by Petsalis, a negative result is reported. In the cases of Louros neither parasite nor pigment was found in the placental blood. On the other hand, in all the cases examined by the author, the hematozoa were always found in the placenta, but without a trace of histologic change, which would indicate that the latter is infrequent. In all cases in which infection of the fetus or newborn ocurred, it is probable that this was endoplacental inoculation following hemorrhage (as though by Bignami and Sereni) rather than physiologic transmission of the parasite.

The transmissibility of the virus alone by way of the placenta is pure theory. Admitting that the virus and the hematozoa pass the placenta, since no Greek or Italian observers have found the latter in the fetal blood, if we attempt thus to explain congenital malarial cachexia postulated by some on clinical experience alone, and if we also interpret thus the opinion of those who believe in congenital malaria and that children of malarial mothers are of smaller weight and size, pale, splenomegalic and anemic, then why have the Greek peoples, among whom malaria is so prevalent, not disappeared? If placental transmission occurs, it is an exceptional occurrence. It is incontestable that some microörganisms do pass the placenta, but it does not follow that this is true of hematozoa. We have observed repeatedly that infants born of cachectic mothers are in excellent health and well nourished. Defenders of the theory of hereditary transmission also admit that the malaria of the mother has no influence upon the health of the fetus.

Isolated findings are not convincing. Bouzain's case is not valuable since the microscopic examination of fetal and maternal blood was made 12 days after confinement. Same is true of Topi's. Bein's case is the only undoubted case in which the hematozoa were found in fetal and maternal blood.

Italian observations on 18 fetuses and our 17 cases showed the parasite in the blood of mothers but not of the fetus or newborn. Three cases are reported by Jansco. Against the one positive finding of Bein, we have therefore 38 negative cases: 18 abortions with dead fetus; 18 full-term, survived; 2 abortions, survived.

Admitting the possibility of transmission of the virus, then:

1. Under what condition does transmission occur?

2. If transmission occurs, what is its frequency? This can only be determined by future investigations based on attentive study of the blood and viscera of the aborted and stillborn fetuses, of their dead mothers, and placentas. The blood of surviving infants, of their mothers and of the placentas must also be examined microscopically. Experience has shown the author that maternal malaria does not influence the general condition of the fetus. In seven mothers with splenomegaly, he found the parasite in three, but in none of the newborn. Examination of the blood of the infant was made from 3 to 30 days after birth.

His conviction of the non-transmissibility is based on a study of 20 cases, in which the blood of the malarial mother, newborn, umbilical cord, all coats of the placenta, the liver and spleen of two dead infants were examined microscopically. Examination of the placenta showed an abundance of parasites undergoing schizogony on the maternal surface of the placenta, but they were either absent or less numerous in the fetal area. The mother's blood showed tertian (six cases), estivo-autumnal (twelve), quartan (one), mixed tertian and estivo-autumnal (one). Author believes the fetal surface of the placenta and the fetal blood contain an antitoxic substance opposing the development of the hematozoa, destroying them in their passage to the external surface of the placenta. This explains the absence of the parasite in the fetus and the innocuity of the virus upon the general condition of the child.

Researches of Ehrlich and Hayem have shown that the blood of the fetus and newborn infant differ in constituency from that of adults.

Density is greatest at birth, diminishing gradually until the second or third month. Hemoglobin index is high in newborn and the first days of life, and reaches its minimum at the third month. Large red corpuscles are most numerous immediately after birth, the small slightly larger than in adults. In the first four days a large number of nucleated red cells are found, sometimes as late as the second month. Microcytes are much less abundant in newborn than in infants. According to Hayem the leukocyte count of newborn is three or four times that of adults, and according to Gundobin, the leukocytosis diminishes by degrees about the fifth day, reaching normal count about the tenth day.

The proportion of white to red is 1:30 at birth, 1:150 to 180 during the first month, 180:210 from first to fifth month (Demme). At birth the blood coagulates more rapidly because of great quantity of fibrin. Do those differences in blood constituents cause the fetus and newborn to react against the infecting agent? Do they produce the immunity to malaria which extends over the first month of life?

Pathology and Pathogenesis.—Autopsies upon cases of malaria are becoming very infrequent in the United States. This is especially true with respect to children. The benign malarias rarely result in death unless they have been neglected or the diagnosis overlooked.

The post-mortem findings can be predicted from the clinical history. If the infection has been very recent not much black pigment has been deposited and the color is darkened merely on account of the venous hyperemia. In unstained sections much golden yellow pigment can be made out. If there has been more prolonged exposure to infection with indifferent treatment and pernicious symptoms supervene to cause death, the organs appear very dark slaty-colored or quite black from deposit of hematin. The black pigment is in the endothelia, mostly in areas of slow current at first, later it is carried deeper into the adjacent cellular tissue. It is most in evidence in the large macrophages of the spleen pulp and bone marrow and in the hepatic vein areas of the liver. The macrophages contain red blood corpuscles, mostly infected, free parasites or their fragments, smaller phagocytes sometimes "to the third degree" (Barker), meaning that the engulfed macrophages may in turn contain phagocytes. Macrophages are best studied in smears from the spleen pulp. If death occurs early the color of the spleen and liver is reddish-brown; if much pigment has been stored it may be quite black. The very slaty **liver** of perniciosa is in strong contrast with the boxwood-colored liver of yellow fever; in fact, it is quite diagnostic. Care must be taken not to confuse post-mortem discoloration with it. The latter is often absent posteriorly, there being a sharp line of demarcation. Cloudy swelling, focal necrosis, round cell infiltration in the portal spaces, presence of black pigment in coarse blocks, and yellow pigment in diffuse patches centrally in the lobules are the chief changes. Later, there is some fibrosis and less pigment; fatty degeneration is infrequent.

The **spleen** in acute malaria is swollen. Its color is from dark-red to black; it is soft in proportion; the very black spleen is diffluent "so soft that the tarry pulp can be washed away by quite a gentle stream of water" (Manson). In such cases very little can be made out even from smears. We encounter focal and diffuse necrosis, round-cell infiltration, proliferation of macrophages, pigmentation; parasites numerous, as a rule. The spleen of chronic malarial cachexia is large, hard, flat, fan-shaped, a grayish-chocolate color and often of enormous size. Here we have, not turgescence, but hypertrophy, with a narrowing of the pulp sinuses, the increase being mostly a connective tissue hyperplasia. The spleen is no longer a habitat for plasmodia.

The spleen of hemoglobinuric fever is not characteristic. It presents the composite picture of more or less chronic malaria, plus acute pernicious fever. Descriptions found in the literature easily correspond to any intermediate condition suggested by history. If death follows partial convalescence, from renal failure, pigment may be entirely absent.

The **kidney** of *falcipara* gives the picture of toxic nephritis. As a rule, there is not much pigmentation. Siderin is found in the peritubular connective tissue, hematin blocks mostly in the glomeruli. Degeneration is confined mainly to the convoluted tubules. The changes of anemia, hydraemia and passive congestion are encountered. The hemoglobinuric kidney shows all the changes in an advanced and aggravated form. The tubules are blocked with debris of blood elements, or the material can be interpreted as fragments of epithelium stained with hematin. The filtered portion appearing in the urine gives the spectra of hemoglobin and methemoglobin.

The **brain** of cerebral malaria is characteristic. It is of a leaden color, the vessels of the pia seem darker, especially in the choroid plexus; we see punctiform hemorrhages; pigmentation follows the distribution described for other organs. There are capillary thrombi inclosing agglutinated infected cells. Degenerative changes are seen.

The **endocardium** may also be pigmented. Degenerative changes are seen in the **muscle.** If death occurs during pyrexia, especially in cerebral cases, the right heart is full of black blood.

Black and yellow pigment can be seen in the **lungs.** The former must not be confused with anthracosis. Passive congestion and focal capillary thromboses may occur. Large infarcts must be due to other causes. In comatose malaria passive congestion is very pronounced.

The **bone marrow** at times shows the picture of toxic anemia, at others, is dark red to black and in the latter case diffluent. Macrophages have been mentioned. The smear is in accord with the major clinical event. The marrow is most probably the principal organ of larvate malaria. The condition of the marrow is reflected in the blood. Justi, quoted by Schilling (Torgau) holds that the return of the neutrophiles to a complete normal ratio is the first evidence of a complete cure and diagnostic of such. He has reference here to the modified Arneth count.

The Blood.—The effect of malarial infection upon the red and white blood corpuscles is quite characteristic. The pathogenesis of malaria, so far as it is known, can be studied tinctorially and chemically in the blood. The alterations in the infected red cells are best studied in preparations stained with slightly alkaline polychrome stains. The red stippling so often encountered in *vivax* (Schüffner's dots) has been the subject of exhaustive studies. A similar stippling can often be seen in "*malariæ*" if the proper technique is followed. Maurer, who first described maculæ in blood of *falcipara*, gives exact directions for bringing these out. He considers them to be edges of naked stroma exposed by surface erosions, made by feeding parasites. Schilling (Torgau) considers them identical with the spherical dots of Schüffner. He thinks we are justified in assuming that they stand in close relationship to the formation of methemoglobin, parallel with the conversion of hemoglobin into the hematin of the parasite pigment.

Alteration in the appearance of the red cells receives interpretation according to the technique employed. Wet-fixed preparations show spherical pale maculæ, giving pictures more or less characteristic for each species. Schilling (Torgau) believes that all changes can be interpreted as an expression of a chemical alteration of the hemoglobin with the dissociation of the albuminous stroma.

The anemia is in excess of that due to loss from infected cells. Most authors agree on this point. Simpson, using a special method of enumeration, has found such ratios as 25:1 and 30:1 to the number of parasites.

Can we demonstrate a malarial toxin? Many unsuccessful attempts to do so are recorded. Such of the positive experiments as are on record were with blood or serum taken at the chill stage during dispersion of merozoits when hematin only is liberated (when we are dealing with used-up corpuscles). Simpson, working with 100 sera of malarial patients, acting upon washed normal and malarial corpuscles obtained hemolysis only seven times, one positive result being with serum of a case of blackwater fever, five from sera with vivax infection during the prodromal stage. Brem obtained hemolysis from parasitic extract and secured hemolysis in 20 minutes. The hemolysin is thermolabile. Zeiler and Brem have also demonstrated an anti-hemolysin in normal serum and that from pernicious malaria. Casagrandi and De Blasi describe a hemolytic toxin which destroys red blood corpuscles. Garbarrini reports the results of experiments in complement fixation in 22 cases of malaria, acute and chronic. "The blood taken either during the rigor or the febrile stage and for a few days after the attack will deviate complement if it has been deprived of its natural hemolytic amboceptors. In latent malaria the same reaction may be obtained, but more feebly. In 'chronic malaria' the reaction is always negative."

Celli's, Mannaberg's, Gualdi and Montesano's negative and Rosenau's positive experiments are well known. Brown, experimenting with hematin from various sources, injected intravenously in quantities estimated to approximate those in malarial fever, invariably obtained reactions in every way similar to those seen clinically in man. He used alkaline solutions and reports the occurrence of phagocytosis and endothelial cell necrosis and other evidences of toxic anemia. Jobling and his co-workers advance the idea that the toxic effect of intravenous injection of inert substances, such as kaolin, is due to protein split products derived from the serum proteins. The kaolin acts as an adsorbing medium for the anti-ferment, bringing about an alteration in the ferment-anti-ferment balance.

Thayer makes the point that the toxic effect upon blood making organs adds to the anemia. We know of the unsupported claim by Rowley-Lawson that migration of parasites from cell to cell, leaving abandoned cells to die, is a cause of anemia. We know that it is stated that much capillary hemorrhage occurs in malaria, and, finally, we are acquainted with the writings of Tomasselli, Koch, v. Diessing, Bass and others concerning the toxic effect of quinin upon erythrocytes. Simpson and Edie's experiments are more definite. They find in malaria a marked but variable increase of urobilin excretions after usual doses of quinin.

This much seems evident: The parasite requires an enzyme to digest the stroma of the erythrocyte, upon which it feeds. This cytolytic substance is responsible for the degenerative changes in the red cells, often resulting in their premature disintegration with the escape of siderin and hemolysin. That an antigenic substance exists is borne out by the clinical history, the spontaneous cures and the relative immunity exhibited by old residents, especially those who in childhood had enlarged spleens. That it may exhibit the phenomena of anaphylaxis shown in hemoglobinuric fever. Its presence is also suggested by the agglutination of red blood corpuscles in pernicious malaria. It is not a free toxin.

Phagocytosis.—The earlier writers noted phagocytosis only of dead or injured cells and of pigment. Thayer describes and pictures phagocytosis in wet films. In the examination of many thousands of stained smears we have never seen phagocytosis of parasites. One can see it in wet mounts that have stood an hour or more. Bass says the leukocytes must be pipetted off from cultures before a second generation can be grown. Phagocytosis is well seen in post-mortem smears from the spleen, where the parasite remnants, red blood corpuscles and pigment are found within macrophages. The immunizing process is probably a complex one in which the work of the macrophages and endothelial cells is of greatest importance in the production of immune bodies.

At certain stages of a malarial attack the increase of parasites after a paroxysm may be less than 10 per cent. of the merozoits produced. That this is not wholly due to simple phagocytosis is shown by the presence of degenerating parasites within red blood corpuscles.

Leukocyte Reaction.—Before the first attack there is a slight premonitory mononucleosis. During the chill there is a short transitory leukocytosis. One of us described this as occurring in the presegmenting stage but lasting until the chill. In the fastigium the fall begins and in the interval day of the intermittents there is a decided leukopenia. This has diagnostic value if the case can be followed in the clinic. A single differential count without reference to time has no great value. Arneth count shows in the interval, "stabkernige Verschiebung" (relative displacement to the left).

David Thomson finds by his thick smear counting method that 10 to 20 million malignant tertians will cause a leukocytosis, whereas, 30 to 40 million will produce a leukopenia. He corroborates other writers in finding the mononuclear percentage to be in inverse ratio to the temperature. He gives charts of "aparasitic relapses" in which a leukocytosis takes the place of a paroxysm. One of us mentioned this as post-malarial leukocytosis in the Memphis Medical Monthly in 1902. This accords with Justi who states that a complete return to normal is the first evidence of cure. In malignant tertian this is never less than nine weeks, and may be several months.

Symptomatology.—General Consideration.—Typical symptoms of the "benign" malarias are splendidly described by Thayer, and well enough given in general textbooks. Estivo-autumnal malaria occurs as a drawn out tertian fit, lasting over 20 hours, usually followed by remittent fever with "biliousness" thereafter. Chills and chilliness were observed by Thayer in 71 per cent. of first paroxysms as against 92 per cent. for tertian and quartan. Quotidian paroxysms are common, especially in children, no matter which species of parasite is present.

Single infections with *vivax* and *malariæ*, even in children, when untreated, give a very characteristic fever course, the fit lasting 8 to 12 hours. Multiple infections are usually recognized by the lack of time relation of the alternate paroxysm. *Vivax* may give two paroxysms in a single day, two on alternate days, one, two, three or four in two days. Malariæ in double groups give febrile attacks two days in succession, followed by an interval day, in triple groups they make daily fevers; soon these become irregular. *Falcipara* may segment in small groups at irregular intervals, giving any sort of fever chart from quotidian to continuous fever. Gametes cause no symptoms.

Mixed Infections.—This term is applied to infection by two or more species. The symptom-complex is that of the *last species introduced.* When paroxysms of quartan give way to the curve of malignant tertian, the quartan is still present, but the falciparum dominates the picture, and *vice versa.* Irregular fevers are always due to prolonged infection. After so many cycles the parasitic groups lose their compactness, and gametes are more in evidence, parallel with vegetative forms.

The clinical variants are shown in the illustrative charts from the Memphis Children's Hospital (Figs. 362–368).

Perniciousness.—For some unaccountable reason this clinical distinction seems to be necessary to make a treatise on malaria complete. It is even more remarkable that American writers should insist upon carrying forward the delusion of a hemorrhagic malarial fever. That we may have a malarial apoplexy is true, but such cases are clinical curiosities.

Hemorrhages may be from the brain, the lungs, the intestinal tract and sometimes there are slight bleedings from the kidneys, paroxysmal, and not associated with alarming symptoms. There is nothing to justify classifying this occasional hemorrhage as a type of malaria.

Falcipara, and rarely the two other species when badly neglected, may tend to mass in certain vascular areas, thus giving symptoms that have received the denomination comatose, algid, choleraic, etc.

More accurately, we distinguish cerebral, gastric, enteric, dysenteric, pulmonary, hepatic, cardiac, etc., localizations, associated, however, with massive general infection. Bass, in making smears of cultures of falcipara, found that after these are half the size or more of the containing cell they smear out at the tail fringe like leukocytes. He regards this as proof that they are thick and tough and consequently block certain capillary areas. Segmentation takes place and "the nearest red blood corpuscles cannot escape and are readily infected." He considers this to be the reason why *falcipara* are more resistant to medication; the areas being blocked, quinin cannot reach them. Perniciousness, according to this conception, is imprisonment of parasites in certain vascular areas.

If falcipara necessarily block capillaries and this constitutes perniciousness, then all *falciparum* infections must be pernicious. Post-mortem evidence shows that death may result from coma without any cerebral infection. On the other hand, we find massing of infected cells in the spleen sinuses, where no blocking can occur except from agglutination. Since severity of disease is a question of degree of infection as well as of species of parasite, no clinical borderline for perniciousness can be made out. *Pernicious malaria is neglected malaria.*

Special Symptoms.—Nervous System.—Almost any disease of the nervous system has its symptomatic mimicry in malaria. Foremost are coma, delirium, convulsions, headache, neuralgia and pains of a neuritic character. There may be chorea and epileptoid attacks. Of all these, convulsions are the most common, and, with the fever, may constitute the sole symptoms. Delirium often accompanies moderate fever.

Digestive Tract.—Vomiting is a very common symptom, especially in infants. There may be much epigastric distress, the tongue has a dirty coat, and the breath is offensive. There may be diarrheal, choleraic or dysenteric symptoms in "pernicious" malaria.

The Kidneys.—The urine has the character of fever urine. During the cold stage a fit of polyuria is usual. Urobilin is generally present. In severe malaria we are apt to have a decided nephritis without much fever, often leading to error in diagnosis. Approximately half the cases of estivo-autumnal fever will show albuminuria.

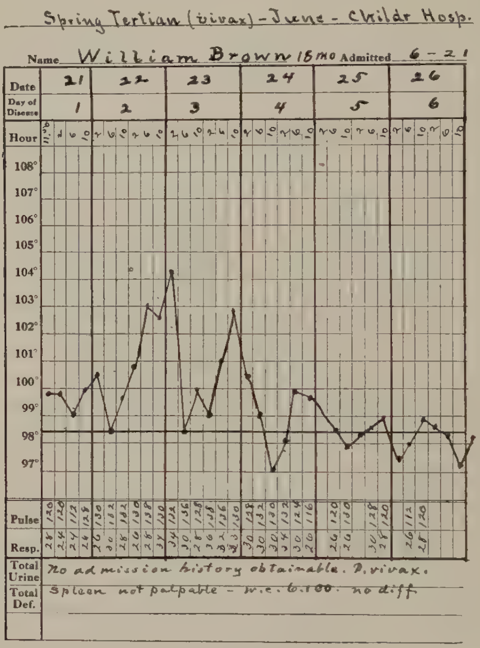
The Lungs.—Respiration is always increased during the fever. Sometimes there is marked pulmonary stasis. Blood spitting may then occur. Marked bronchial irritation occurs in pulmonary localization of parasites.

The Skin.—There is "goose skin" during the chilliness. Miliaria accompany the sweat. There is always a slight icterus. Sometimes, this is quite pronounced. In hemoglobinuric fever the patient presents the appearance of having been coated with ochre. Urticaria is not uncommon, especially after quinin. Rarely, it is paroxysmal and replaces the fever. Herpes of the nares and lips are very frequent. "Fever blisters" are looked upon as a sign of convalescence; they are *not* evidence of blood sterility.

Marked bone-marrow pathology often gives rise to localized pains. Malarial "rheumatism" is not rare.

Circulatory System.—The heart is not often seriously involved. The pulse is always accelerated, and rises and falls with the fever. This is a striking differential sign against typhoid and yellow fever. The character of the pulse varies with the stage of the fever. At first small and of high tension, it becomes full and bounding toward the end of the paroxysm. Faint systolic murmurs may be heard at times. Hemic murmurs are to be expected in grave anemia.

The **special senses** are impaired from quinine intoxication. Malarial amaurosis has been reported. The production of blindness and deafness by the heroic use of quinine is not justified.



Spring Tertian (vivax) - June - Childr Hosp.

Name William Brown 18 mo Admitted 6-21

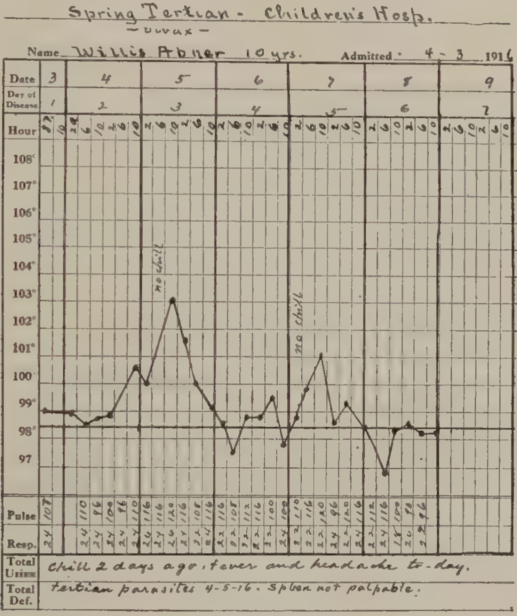
No admission history obtainable. P. vivax.

Spleen not palpable - w.c. 6.100. no diff.

Fig. 362.

Degree of Infection in Children.—Cardamatis says: "Acute and chronic; oftener chronic because of want of early diagnosis, and of failure in therapy. The child is subject to all types and forms. Predisposition to one or another of the three species of the hematozoa is not apparent. Of the 360 cases studied by Concetti and Valagussa, 52 were estivo-autumnal of which 12 pernicious, 85 were quartan, 205 were vernal, and 18 types not defined. Of 838 cases from 0 to 16 years in which author examined the blood he found 179 infants from 0 to 2 years (63 with benign tertian (*vivax*), 106 grave estivo-autumnal (*falciparum*), 3 quartan (*malariæ*) and 7 *mixed* infections). Of these 838 cases by ages, 87 were infants, 92, 64, 71, 71, 45, 70, at two to seven years respectively; 64, 27, 39, 29, 48 from 8 to 12 years, and progressively less for subsequent years. Forms by all ages: Vivax, 274, falciparum 458, malariæ 74, mixed 32. We claim the child has a resistance to infection because we observed some children with schizogonic forms of quartan fever, more rarely those of benign tertian, but with no febrile reaction. Moreover we found the hematozoa in peripheral blood in perfect health.

There were recorded 8 cases, from 35 days to 12 years of age, some with schizogonia of estivo-autumnal fever, as well as quartan; all well-nourished and in good health. Again, we found hematozoa in the blood with temperature ranging from 37.2 to 37.7 degrees, most of them in good health, others extremely afflicted, pale, anemic, feeble. Concetti and Valagussa have made similar observations."


Spring Tertian - Children's Hosp.
— vivax —

Name Willis Abner 10 yrs. Admitted 4 - 3 1916

Total Urine: Chill 2 days ago, fever and headache to-day.

Total Def.: tertian parasites 4-5-16. Spleen not palpable.

FIG. 363.

Bignami observed the same among adults in mild infection of quartan, and says:

1. "Infants and children develop an organic reaction, sometimes feeble, at others strong, against the toxic products of hematozoa; the natural immunity varies in degree.

2. "In Italy we encountered the same relative immunity as among the natives of tropical Africa, but in slightly less degree.

3. "The form of malaria which develops immunity does not always produce anemia, as claimed by Plehn.

4. "The immunity observed by us was not in children with previous attacks, and therefore, differs from that of Koch, Plehn and Firket, and from that observed by Celli."

Acute Malaria.—It is the common opinion of authorities that the smaller the infant the greater the variation of the febrile paroxysms from the classic forms. Bouchut, Valleix and Griesinger directed attention to intermittent fever in infants, characterized by:

Prodromata.—The child is restless, alternately pale and flushed, appears languid, depressed and wretched, yawns, takes the breast without feeding, and between times is nauseated, and vomits slimy matter.

Paroxysmal Period.—All observers note that one of the three stages is absent, either the chills or the sweat. Mariottini in 120 cases of malaria in infants reports 53 without chills.

Chills.—Chills are as sharp as in adults; even if the child does not shiver, the chill will cause it to pale; the nose, face and skin are icy cold. Chills are accentuated in children of later years, in fact, they shiver and moan, some even crying during this stage. The nails, tip of the nose and lips become dark, and a slight redness of the face following pallor heralds the rise in temperature. During this period or rather at the onset of the paroxysm two symptoms predominate: Vomiting and convulsions.

A tremor of the extremities, spasmodic contraction, and tremor of the eye muscles have been observed.

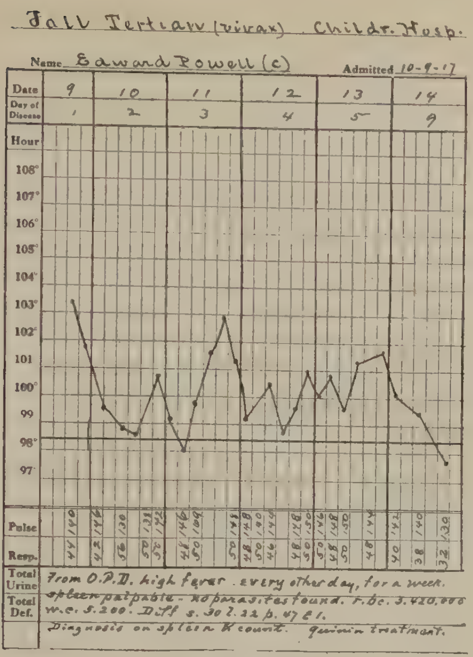
The duration of chills varies from a few moments to about an hour; at other times, the fever sets in little by little without chill; in some, convulsions occur during the febrile period.

Fever.—This lasts from three to six hours, sometimes longer. In infants infected for the first time, fever lasts for long periods, and in exceptional cases, is continuous. In the last group diarrhea may develop, and unless microscopic examination is made the true cause easily escapes detection.

Sweat.—This is entirely absent in infants; it is replaced by slight moisture following dryness of skin; at times the head and arms only are slightly moist; at others, only the nape of the neck. After a paroxysm the infant again becomes lively, but if paroxysms recur the child becomes anemic, peevish and exhausted from diarrhea.

Spleen.—In the first paroxysm, there is no splenic hypertrophy, but some tenderness may be elicited on pressure. Splenomegaly is one of the principal indications of malaria in children. Referred pains at the base of the thorax with difficult respiration may lead to diagnosis of pleural or pulmonary disease.

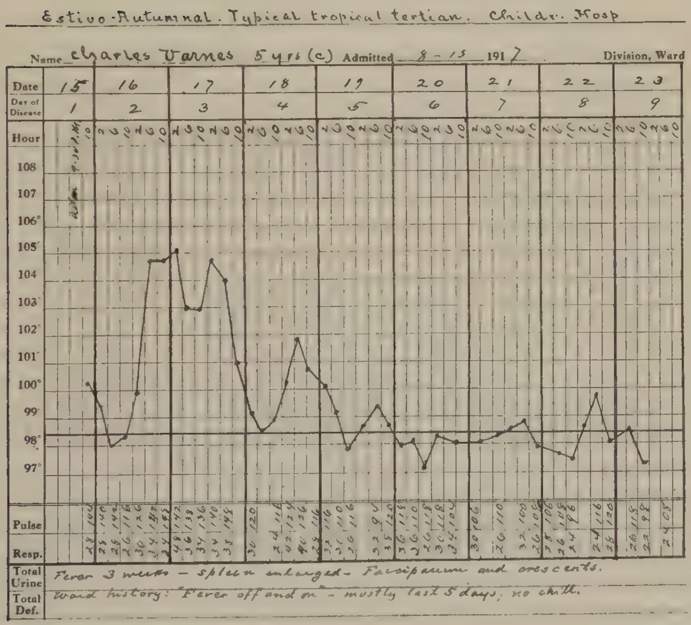
Form of Fever.—Quotidian or irregular fever is commonest in infants. If the infection be a bit intense, fever will be remittent or continuous. Although an infant may be infected by frank quartan, this is rarely manifested clinically (see exception, on chart). The quartan form may assume the quotidian, tertian or irregular periodicity. Statistics based on clinical observations only rarely include quartan, but it is not so rarely found in statistics based on microscopic findings. Of 1087 cases under 16 years, 554 were quotidian, 203 tertian, 4 quartan, 216 continuous, 47 irregular, and 63 unclassed.


Fall Tertian (vivax). Childr. Hosp.

Name Edward Powell (c) Admitted 10-9-17

From O.P.D. high fever every other day, for a week.
~~spleen palpable~~ - no parasites found. r.bc. 3.420,000
w.c. 5.200. Diff s. 30 l. 22 p. 47 e 1.
Diagnosis on spleen & count. Quinin treatment.

FIG. 364.


Estivo-Autumnal. Typical tropical tertian. Childr. Hosp

Name Charles Varnes 5 yrs (c) Admitted 8-15 1917 Division, Ward

Fever 3 weeks — spleen enlarged — Falciparum and crescents.
Ward history: "Fever off and on" — mostly last 5 days; no chill.

FIG. 365.

Later Childhood.—The older the child the more the febrile paroxysms approach the type seen in adults. From the ages two to six years the paroxysms are more characteristic, chills last from one-fourth to one-half hour, and febrile and sweating periods are more marked. Jules Simon says that fever is generally simple or double quotidian or tertian.

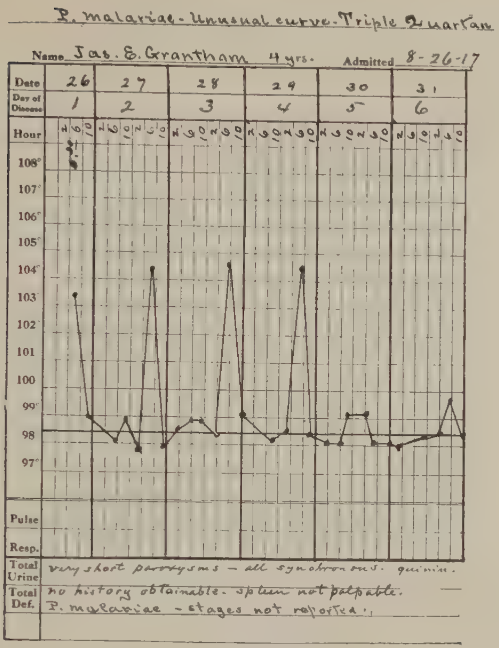
In infants under two years the most frequent complications are gastro-enteric disturbances, at later years convulsions (with or without delirium according to age). The delirium, together with constipation which generally replaces the diarrhea seen in early life may lead to erroneous diagnosis of meningitis. If microscopic examination is impossible, the history, season and spleen examinations are great aids. Our statistics show the ages of two to seven the most susceptible years, and in these we find splenomegaly most often. Some authorities claim that splenomegaly is not noted in infants, but in cases examined by the author, in which hypertrophy was found, 75 per cent. showed hematozoa in the blood. Of 211 children under 12 years of age, attended during an epidemic, 81 had splenomegaly, 69 of these showed the parasite. Of 80 other children with splenomegaly, 64 showed the parasite. Of other series of 29 with splenomegaly, 21 showed hematozoa (total 200 with splenomegaly, 154 carriers, 77 per cent.). Wlassiewsky found the proportion to be 72.1 per cent. Stephens and Christopher conclude:

1. In early life (0 to 2 years) the number of infected infants is in excess of the number with splenomegaly.
2. After the second year the number of cases of splenomegaly is slightly in excess of the number of the cases of infection.
3. After the tenth year the number with splenomegaly is generally large in proportion to the number of infected children.

Types in Later Childhood.—The most frequent types met with are quotidian; tertian, continuous, and, more rarely quartan. However, continuous fever is more common in this period of life than in earlier years. Of 500 cases of continuous fever we found 214 children (5 under 1 year, 58 from 1 to 5 years, 83 from 5 to 10 and 68 from 10 to 15). All these cases had the aspect of the remittent and continuous biliary fever of the tropics (bilious vomiting, jaundice, diarrhea, etc.).

Chronic Malaria.—Is more common in children than acute. Of 360 cases Concetti found 258 chronic. The author found in rural countries 75 per cent. children had chronic 554 out of 758 cases at Marathon 73.08 per cent. 160 well; 44 acute malaria; 302 had splenomegaly.

Cachexia.—The first and most important symptom of chronic malaria is extreme anemia, the sign of the onset of cachexia. Repeated paroxysms, due to faulty or incomplete diagnosis are the cause of chronicity. We found among 641 children under 12 years nine with splenomegaly and cachexia, without any paroxysms and with

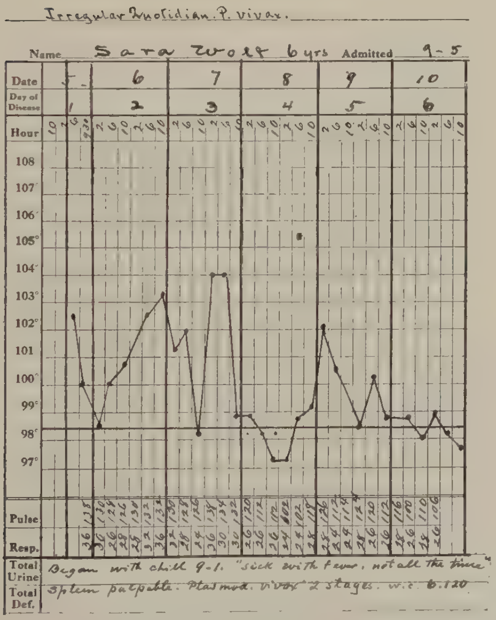


very short paroxysms — all synchronous. quinine.

no history obtainable. spleen not palpable.

P. malariae - stages not reported.

FIG. 366.



Began with chill 9-1. "sick with fever, not all the time"

Spleen palpable. Plasmod. vivax 2 stages. w.c. 6.120

FIG. 367.

no evidence of other diseases. In these cases the cachexia evidently developed insidiously and gradually without repeated paroxysms.

Symptoms of Cachexia.—Pallor, exhaustion, wasting. In infants a peculiar facial appearance, waxy, unlike anemia of other diseases, and differing also from appearance in older children and in adults. Splenomegaly is a characteristic and essential symptom. If untreated, febrile attacks produce malnutrition, wasting, muscular flabbiness, and extreme anemia (from two to one million red cells or less per cubic millimeter). After febrile attacks, progressive enlargement of the

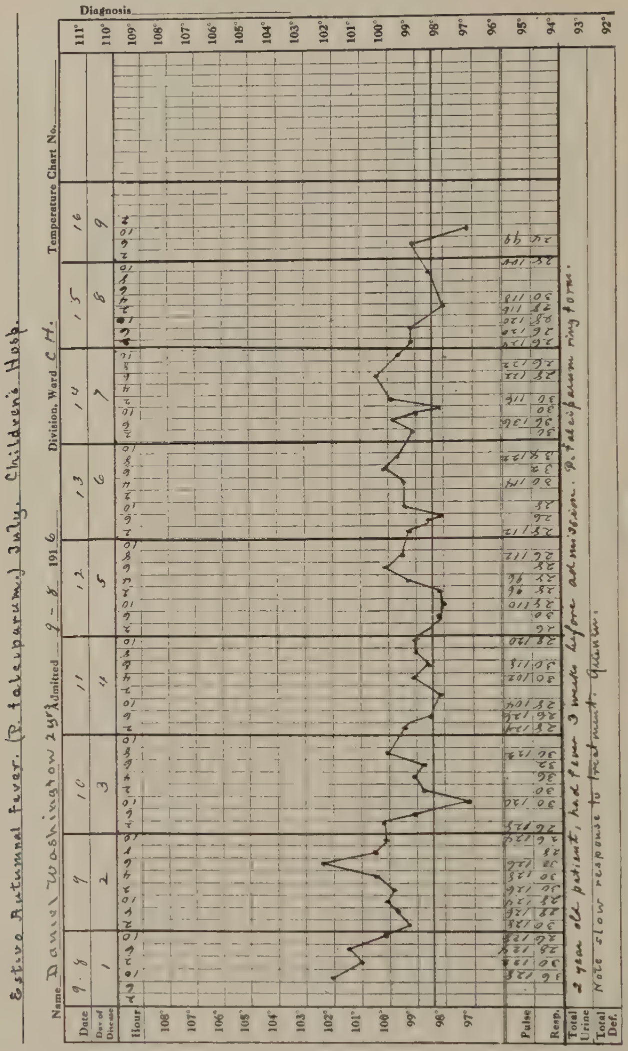


FIG. 368.

spleen, especially in children over two years, sometimes gigantic spleen reaching almost to the pubic symphysis. The parenchyma becomes hard, and the great hypertrophy may produce complications such as dyspnea by pressure on the diaphragm.

Frequency of Cachexia.—Unfortunately the disease is very prevalent in this form in Greece. Of 140 children from 4 to 12 years, 64 had splenomegaly.

Cachexia without Splenomegaly.—The degree of hypertrophy is not an index of the degree of cachexia; in fact, we have encountered children with extreme cachexia and hydremia, with only slight splenic hypertrophy. Of 108 cases (under 12 years) with severe cachexia and hydremia, 83 had splenomegaly, while 25 had only moderate hypertrophy.

Hemoglobinuric (Blackwater) Fever.—"Daniels saw a case in a half-caste about five years old; Wendland, van der Scheer and Karamitsas observed cases in children of (as young as) four; Lipari mentions two cases in children of three; Masterman in a girl of two, and Oetker one in a two year old child; Fisch saw cases in children of 14 months and 2½ years, and McElroy one at 12 months" (Deaderick and Thompson).

One of us saw a child of 18 months, a boy aged six years and one of eight years. The latter recovered. All were white.

Symptoms.—There is almost always a history of previous more or less frank malaria. It is unknown in new-comers, in strong contrast to "pernicious" malaria. From the Canal Zone one was reported as having been exposed only two months, a mild case, following a thorough wetting. All tropical writers agree that cases under six months' residence are very rare.

The first symptom, as a rule, is the chill. This will vary, of course, with age of the child. It will probably be described as "different from the other chills." Then, after some vesical tenesmus characteristic urine will be voided. Urination may be scant and frequent, or copious, and the color will be from red to almost black. There is a heavy albumin content. In a few hours an intense jaundice appears, generally coincident with copious vomiting of spinach-green material. The stools are black, tarry, and tend to become loose even if the doctor with his calomel does not get there first. Gradually the serous diarrhea will cause the stools to pass in color through deep green to yellow. The pulse is "gaseous," the breath labored, the dyspnea being due to loss of blood. There is marked anxiousness and great nervousness. Hiccough occurs very often, and is considered an unfavorable sign.

Death on the first day is due to coma. Later it is from suppression of the urine or extreme anemia. Occasionally, there is sudden death during convalescence, probably from embolism. The malarial infection is never a major event unless the fever is paroxysmal (intermittent). Only rarely are parasites found after the first acute hemolysis. The infected corpuscles evidently dissolve first. Occasionally, one sees cases that have become sensitized to quinin. This never occurs except in the presence of malarial infection. Persistence in quinin therapy will promptly kill such patients.

Pathogenesis.—There is so much difference of opinion and so little real scientific evidence that any discussion further than indicated under "pathology" would be a waste of space.

Treatment.—Cases of hemoglobinuric fever never require quinin until three or four days after the onset. A test dose may then be given to see if hemolysis results. Recrudescence of the residual infection is not uncommon.

The vomiting is often allayed by copious drinks of hot water. Iced carbonated drinks may then be tried. Effervescent alkaline drinks are much in favor. In their absence, frequent small doses of sodium bicarbonate are useful. Morphin should be absolutely prohibited; the consequent suppression of the urine is almost always fatal.

All other treatment must be symptomatic. Enemata or the Murphy drip may keep the kidneys at work, especially if much fluid has been lost from vomiting and purging. Transfusion should be tried in extreme anemia; hemoglobinuric blood is hypertonic. Convalescents must be kept under care until the blood is restored and sterilized.

The prognosis in children is very grave. On account of the small number of cases recorded it is difficult to give figures.

Prognosis of Malaria.—Regarding the prognosis of malaria in children in general, the results depend so much upon local conditions and prompt diagnosis, that all one can say is that malarial infection becomes grave according to the length of time it has run before any diagnosis has been made. There should be no mortality, except in falciparum infection, and then only if valuable time has been lost. From 1 to 3 per cent. of children with falciparum infection, die. Infants are not very resistant to sequelæ, especially in hot weather.

Some authors attempt to classify complications. These are never anything but intercurrent diseases for which the malarial infection is not responsible.

Diagnosis of Malarial Infection.—A frank tertian or quartan intermittent is rarely anything but malaria. Quotidian, subtertian, remittent and continuous fever is not diagnostic of any disease. In infants and very young children the temperature curve is not often diagnostic. In hospital practice we rarely see typical periodic charts, because, as a rule, the diagnosis has been established, either hematologically or presumptively, and treatment has obliterated the character of the curve.

In pediatric practice the diagnostic methods are: (1) Presumption of infection from the history. (2) Study of the symptoms, i.e., the onset, fever, pulse and temperature ratio, physical examination, etc.

(3) Finding the parasites in the blood. (4) The "therapeutic test"—the leukocyte variation in very young children may lead into error. The variation of leukocytes is discussed under "Examination of the Blood." The specific diagnosis is given third because this is the order in which the technique of examination puts it. It is, of course, the only real diagnosis.

Typhoid fever in the first week can be distinguished by the positive blood culture, culture of the stool, the dry skin, the diazo reaction, the absence of parasites in the blood (doubtful value) but more especially by the lower pulse rate.

The cachexia of malaria is distinguished from ancylostomiasis by the large spleen which is absent in the latter. Ashford says the hookworm spleen is contracted. Leishmaniasis and other tropical diseases may give trouble and require laboratory methods for differentiation.

Liver abscess may come into question in older children. The Schilling count would eliminate it from consideration. Sepsis, malignant endocarditis, pneumonia, cerebrospinal fever, intestinal diseases have a frank leukocytosis. Influenza would give catarrhal symptoms, and have a different seasonal incidence, as a rule.

Our records show a leukocyte average of 6000 for malaria, though very high counts are sometimes seen. Algid malaria might simulate "ptomain poisoning," both having high counts. The former does not give the great abdominal tenderness, comes on more gradually, and parasites are always found.

The gastric disturbances of malaria are confusing. Often there are no other symptoms.

The fevers of syphilis and tuberculosis must be considered.

We have not found the spleen enlarged very often in acute malariæ in the wards of the Memphis Children's Hospital, although as a rule it is tender on pressure.

Finding the Parasites.—In vivax and malariæ the plasmodia can be found if there has been no quinin "tinkering." The schizonts of these species pass a large part of their cycle in the general circulation. In falciparum infection the parasites multiply rapidly and, when symptoms are urgent, we can with patient search find the rings in well stained specimens. Unfortunately, too often the diagnosis has been prejudiced by quinin. Enormous doses of this drug depress the thermic centers, and any fever will yield to it for a time. In the absence of previous therapy we ought to find the "benign" types in 75 per cent. of clinical cases. P. falciparum is perhaps not found in over 30 per cent. of first examinations, unless the concentration method is used. It has been held that falcipara sometimes get into the peripheral blood at night. We have corroborated this, perhaps as a coincidence, a few times.

For further data, see "Degree of Infection," p. 756.

Examination of the Blood.—Three methods can be recommended: The stained ordinary smear, which also serves for the differential count;

the thick film of Ross or its modifications; the centrifuge concentration method. Fresh, unstained smears present too many pitfalls for the beginner. They are suitable only for vital study, which is not necessary for differential diagnosis. The thick film also presents many confusing pictures for any one not very familiar with it.

Method of Obtaining the Blood.—Having provided several very clean slides (must be total absence of grease or soap) obtain the blood by puncturing the ear or finger, previously rubbed off with a pledget of cotton wet with gasoline. Wipe off the first drop or two, being careful to get the surface completely dry so that the next drop will not spread by capillarity. Take up a slide by its lateral edges, and, having just heaped up a fresh drop, touch it with the slide without mashing the drop. The position of the drop and the manner of spreading it are shown in the diagram (Fig. 369). A Wright spreader ¾ in. wide prevents the smear from spreading to the edge. If the blood is watery, hold the blood slide vertically and trail the blood upwards. In either case, agitate the slide rapidly in the air so as to dry it before crenation or other artefacts can form. In very sultry weather the slide should first be warmed. The film must be protected from flies. A correct smear has the outline shown in Fig. 369. It should not reach either sides or end and should be thin enough not to show too much overlapping. Always get the blood smeared out immediately after it is

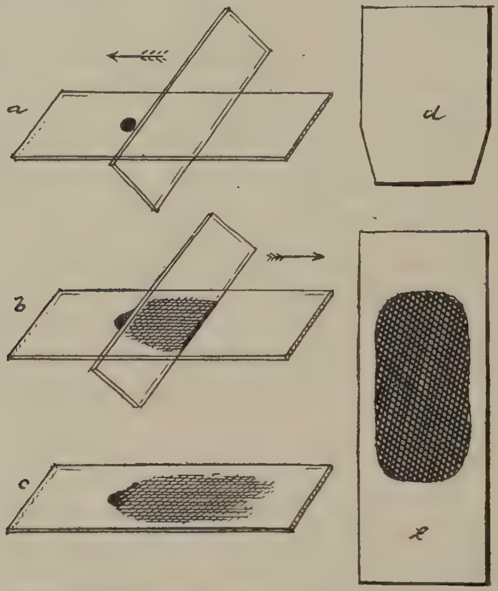


Fig. 369.—Making the trailing smear. N.B. This method does not injure cells and carries less large cells to the end. *a*, Approach. *b*, Trailing the blood angle 35–70°, according to the viscosity of the blood. *c*, Proper distribution. *d*, Wright spreader. *e*, Thick film. N.B. Must be even and thin enough to dry without cracking or blistering.

expressed and dry it rapidly. The right size will come with very little practice. These precise directions must be followed for successful work.

A thick film is made by obtaining three such drops in a transverse row and then dragging out the smear with the other slide by a quick jerk. Such a smear is very much more satisfactory than the "puddle smear." Thick films must be carefully dried or the subsequent steps will fail. The preparation should be warmed before decolorizing (see below).

The concentration method worked out by Dr. J. S. Fleming has served us well. It can be used for field work or visiting practice by keeping on hand the vacuum ampules* containing 2 per cent. sodium citrate, should be 6.6 per cent. salt and 0.1 per cent. formaldehyde. With the attached needle blood is obtained as with the Keidel tube for serological work. The blood so obtained is good for 12 to 24 hours. After that the red cells no longer hemolyze. The principle is that a state of fixation is reached at which water will still hemolyze the red cells. A mixture of the above proportions can be kept in small vials, and blood obtained by vein puncture with a Luer syringe. A convenient dilution may be made in the proportion of 2 c.c. of blood to every 8 c.c. of the solution.

The time of fixation may be shortened by the addition of five drops of 1:10 dilution of formalin. Centrifuge the contents of the Keidel tube or pour from the improvised container into a centrifuge tube. For the larger Keidel tube a large centrifuge shield is needed such as the 50 c.c. Cornell holder. The supernatant fluid is poured off, water added and the mixture shaken or stirred until all red cells are dissolved. A second centrifugation will leave a very small residue consisting of leukocytes and parasites. Drain the tube well, collect the sediment with a capillary tube, and spread it on a slide, dry well short of coagulating heat. It is then ready for staining.

A very large experience has convinced us not to pin our faith on one-solution stains. The Giemsa one-solution stain keeps better, evidently owing to the glycerine it contains. During the summer of 1905 one of us worked out a proportion of Azur and eosin which has served this writer in many thousand smears. A large number of practitioners are now using this method to the exclusion of all others.

Provide good fresh distilled water. First test the water as follows: With clean forceps put a crystal of hematoxylin into a chemically cleaned test tube; pour in a convenient quantity of water and shake during five minutes. The faintly yellowish solution should become violet blue in from one to five minutes.

Pure methyl alcohol; this is often too acid. If it seems to give trouble by giving too acid a differentiation, it may be re-distilled or shaken with a trace of sodium bicarbonate.

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* Furnished by Hynson, Westcott and Dunning.

A 1 per cent. aqueous solution of eosin, yellowish.

A 1 per cent. solution of Azur II in water.

Pure aceton.

A shallow Petri dish, or the author's aluminum staining dish.

Technique.—With a pipet overlay the blood film with methyl alcohol—one minute.

Into 5 c.c. distilled water drop two drops of the eosin, shake, add four drops Azur, shake, pour into dish. Place a fragment of glass into Petri dish as a shelf to rest the slide on, and after fixation invert the slide while wet, face down into the solution. The aluminum dish has cleats for supporting the slide and will hold three slides for which the amount of stain just suffices.

Stain for 10 minutes. Rinse off with distilled water, mop off with a pledget of cotton, dripping wet with aceton. Don't rub! This removes precipitates and dries the film instantly.

Examine with low power. An ideal polychrome picture should result. If the leukocytes are not very deeply stained there is no use searching for parasites. Either the proportion of Azur was insufficient or the mixture was too acid. If the mixture in a glass dish over white paper is not entirely blue, dilute the eosin with sufficient water to bring about the proper balance. If this is not the fault, the mixture is acid, either from the water or alcohol. Almost always, if the stain is of good quality and the water stands the Giemsa test, there is no trouble except from lack of attention to detail. If the red cells are blue or slate colored, leave the slide in distilled water for half to one minute.

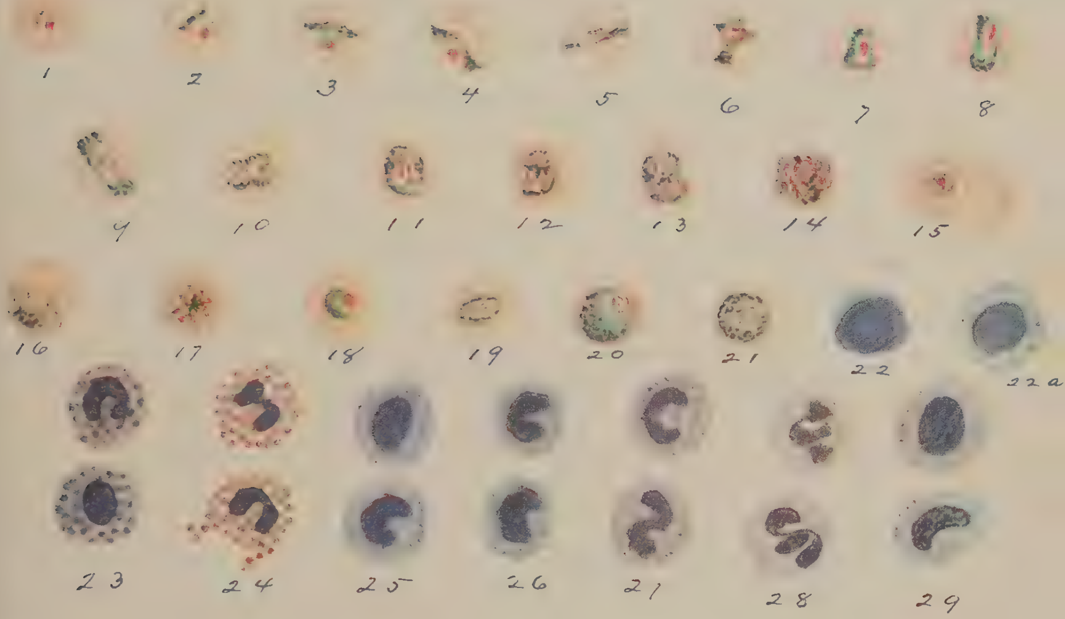
For the thick film a satisfactory plan is the following. First, dry the film for one hour in the incubator. Fix with methyl alcohol containing 1 per cent. hydrochloric acid for three minutes. This should be changed several times. Wash in a gentle stream of water for five minutes. Stain for 15 to 20 minutes. Dry same as thin smear. The concentration film is fixed in neutral methyl alcohol two minutes, stained for 20 minutes, dried with aceton after washing.

Parasites more than half grown and gametes can be picked out with the low power (16 mm.), using however, a 10x ocular. The writer uses a 1.8 fluorite immersion lens with a x12.5 ocular and a wide angle triple condenser. This is ample definition for studying the finer structure of the parasites.

General Morphology.—Two main forms are recognized; the schizonts in various stages, and the gametes, which should also be recognized before they are grown because they are then still vulnerable to quinin. In both forms we must recognize the characteristic staining reaction. The parasitic body is robin's egg blue or paler, sometimes greenish from admixture of fine pigment. The chromatin* is from pale pink to deep carmine red. No matter how fine the chromatin, it is never yellowish or purple. In fact, there is nothing purple about a malaria parasite when stained with polychrome. "Quinin

* Chromatin = nuclear substance.

PLATE II



Parasite of quartan malaria (P. malariæ). Leukocytes according to Schilling-Torgau.

1–14.—Developing schizonts.

16, 17.—Segmenting bodies; 16 polar segmentation.

4.—Polar schizont.

5, 8, 9.—Equatorial band form. Not necessarily confined to species.

15.—Young microgametocyte.

19, 21.—Microgametocytes.

18, 20.—Macrogamete.

Note compact form throughout, deeply staining cell and parasite, black, coarse pigment, small segmenting body, polar chromatin of macrogamete, loose and more abundant chromatin in microgamete.

23.—Group I; basophiles, normally under 1 per cent.

24.—Group II; eosinophiles, normally 2 to 4 per cent.

<table>
<tr><td>25.</td><td>Group III</td><td rowspan="4">neutrophil</td><td>myelocytes, normally 0 per cent.</td><td rowspan="4">total 67 per cent.</td></tr>
<tr><td>26.</td><td>Group IV</td><td>metamyelocytes, normally 0 per cent.</td></tr>
<tr><td>27.</td><td>Group V</td><td>stabkernige, normally 4 per cent.</td></tr>
<tr><td>28.</td><td>Group VI</td><td>segmentkernige, normally 63 per cent.</td></tr>
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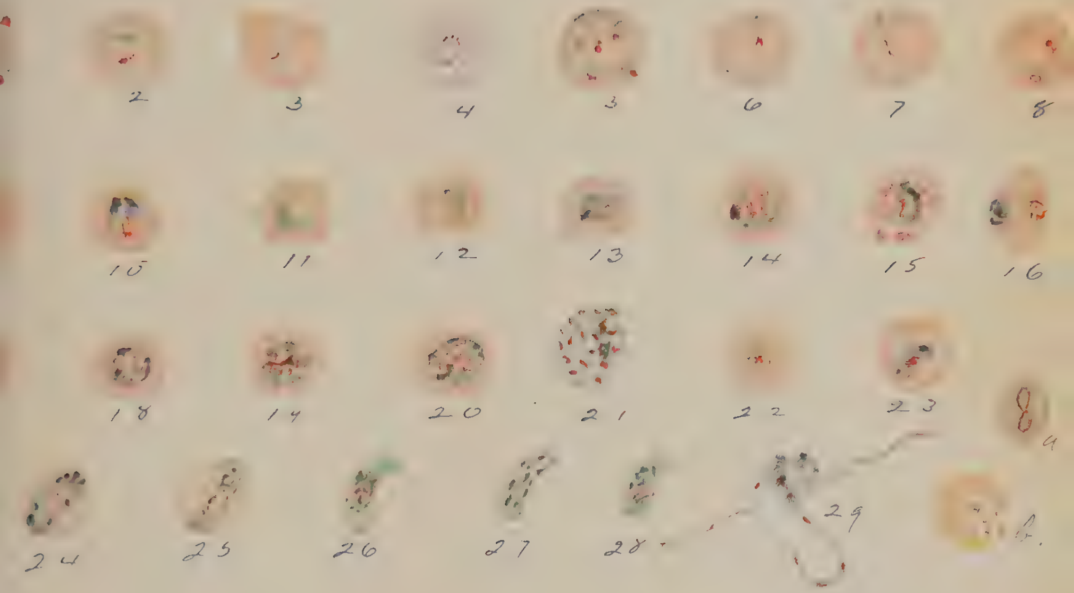
22, 22*a*.—Group VII, lymphocytes, normally 23 per cent.

29.—Group VII, large mononuclear and transitional.

PLATE IV



Parasite of estivo-autumnal malaria (P. falciparum).
(Parasite of tropical fever, malignant tertian, subtertian, remittent and continued fever, "pernicious malaria."

1 to 21.—Developing schizonts. 1 contains 3 parasites, one on edge, two showing the mound on which the nucleus sits, giving a thickening or signet ring form.

2, 4, 5, 6, 11, 12, 13.—Basophilic stippling, not observed in "benign."

4, 5.—Showing greatest anisochromasia.

7, 16.—[illegible] bands, resembling "malarine," which compare.

8.—Large ring, characteristic of species, 2 Maurer maculæ shown.

15.—Just passing from ring stage, marked "perniziosafleckung."

17.—Double chromatin, Ewing's "conjugation."

11, 12, 13.—Involution forms, necrotic cells.

9, 10, 11, 12, 13.—Effect upon r. b. c., shrinking of cytoplasm, basophilia.

19.—Small segementing body; so-called plasmodium præcox.

18, 20.—Presegmenting.

21.—Segmentation; note smaller merozoits than in other plates.

22, 23.—Young gametes.

24, 26, 28.—Macrogametes, broader, chromatin and pigment in center.

25, 27.—Microgametocytes, slender, diffuse.

29.—Exflagellation; not seen in freshly shed blood. Camera lucida: obj. fluorite 1.8 mm; ocular X12, 5.

a.—Cabot loop; *b.*—Platelets in red blood-corpuscle.

forms" often lack the chromatin and may be slightly violet blue. They are usually distorted. Be careful not to mistake platelets for parasites. (See Plates II, III and IV.)

Species Differences.—*P. vivax* causes the containing cell to gradually enlarge and become correspondingly paler. Before segmentation the cell is twice its normal size, and only well-stained specimens show a clear outline. In the parasite, even when very small, the ring is heavier at the pole distal to the nucleus. The nucleus is large, round, carmine red, lying in an achromatic zone and shows no tendency to break up until pigment granules appear. The thick portion of the ring rapidly becomes thicker and turban-like and soon shows several small pigment granules; the granules of vivax always remain so small that they appear brown in color. After 24 hours the parasite is half the size of the containing cell, which has grown in the meantime. During the middle 24 hours it presents the most bizarre shapes with secondary loops and pseudopodia and is now quite characteristic. At this stage the parasites are in the peripheral blood in greatest number. The chromatin has become divided into many small granules, which 36 hours after invasion show a tendency to aggregate into separate masses or clumps. Distinct mitotic figures are seen in 42 hours and the presegmenting stage is now well established. Radial striations are rarely seen. The developing merozoits begin to arrange themselves into a double wreath, and the pigment is pushed to the center. Number of merozoits from 16 to 22.

Plasmodium malariæ is smaller in all stages, more compact, and consequently stains more deeply blue. Its containing cell tends to shrink and become deeper in color. Very blue parasites in very deep orange cells are characteristic for this species. Often they develop from the edge, giving fan-shaped figures. More often they extend across the cell in band form. Even though the cell shrinks, the parasite never quite fills it. The pigment is in coarse black blocks. All stages of the parasite are easily seen during the whole 72 hours. Radial striation before segmentation is well marked, the segments arrange in a single row, the pigment in the center. Number of segments 8 to 12.

The *plasmodium falciparum* is not so easily found in the peripheral blood. During the first 12 hours it feeds upon the surface of the cell, and for a long time clings to the very edge, the chromatin extending out free from the edge. The parasite is thickened at the chromatin pole, giving the typical signet ring shape. It often extends across the middle of the cell in a double band when small. Chromatin divides early. Protoplasmic loop very slender and almost invisible, and hardly thicker at pole opposite the nucleus, even when relatively large. It must be well stained to be seen at all. Retains ring shape until it sinks into cell and then generally leaves the peripheral blood. We owe most of our knowledge of the morphology of the later stages to Bass, and Thomson, from their pictures of cultures. The parasite remains compact throughout, usually showing a single block of black pigment. The segments number 5 to 34. One cannot escape the conviction that the larger number represents numerous or at least double infection of the same cell.

Gametes.—The macrogamete has a clearly defined, compact nucleus, which in the youngest stage is central. The smaller *malariæ* and larger *vivax* gametes are spherical with an excentric nucleus, the *falciparum* being at first round, then oval, and when full grown crescentic, the nucleus always in the center.

The microgametocytes correspond in general morphology with the respective macrogamete, except that the cytoplasm is uniformly paler, more greenish and the nucleus very loose in structure. Both chromatin and pigment are more scattered. The nuclear portion is not excentric.

The Leukocytes.—Basophilic marginal stippling is common in lymphocytes, more so in the large mononuclear cells. Some of these are as large as endothelia. The cytoplasm stains more deeply than that of large lymphocytes. The neutrophiles show marked change of proportion in the sense of Arneth. Youthful transitional cells are much in evidence. Younger neutrophiles are also much increased at the expense of these with segmented nuclei. Eosinophiles are diminished at first, followed by high normal counts.

Treatment.—Prevention.—First we must recall what has been said about breeding places and their abatement. Infants can be protected from bites of insects. Larger children are not so easily controlled. In very malarious regions it is practically impossible to prevent malarial infection, but sickness can be prevented by quinin "prophylaxis." In practice it simply holds down the infection. Stott gave quinin to 3931, 15 gr. three times weekly for five months and 10 gr. similarly the rest of the year. To 3906 he did not give quinin "prophylactically." The first group had 43.3 per 1000 primary admissions, the other had 45.8 per 1000. Linnell used quinin prophylaxis among 2000 coolies with discouraging results. It seemed to act as a slow poison (Stitt). The experience is that as soon as the quinin is stopped, say, in less than a week, cases of malaria begin to appear. In less intensely malarious districts quinin is of benefit. It is very satisfactory for occasional exposures. Bass, in conjunction with the International Sanitary Commission, is trying out systematic cinchonization for 28 days each year. He gives 30 gr. daily to adults, and thinks it suppresses the infection at the source. Protection by screen is peculiarly difficult in the presence of small children. Breeding place treatment offers most where density of population is great enough to justify it. There is no excuse for the presence of anophelines in the center of large cities. Generally a combination of methods must be employed.

Screening.—Stock screens are fly screens, and are worse than useless as a protection against mosquitos; they simply serve to collect them in the house. Contrary to popular belief, mosquitos endeavor to enter the smallest crevice they can force themselves through. As Surgeon White says: "The lean and hungry mosquito can get in but the blood-filled intruder cannot escape." Any crack, crevice or spread in the mesh will admit anophelines.

Three kinds of wire cloth are serviceable. (1) Copper bronze. It should be 18-mesh and should not contain over 1/2 per cent. of iron. It must not be machine-stretched when applied. Contrary to belief, again, it admits more air than looser meshed thick wire. (2) Heavily galvanized wire. This may be 16-mesh. It lasts about three seasons, or less in a damp climate. After being painted it is too dense for comfort. (3) Steel cloth, galvanized and enameled; must not be confused with common black painted wire which is practically worthless and is not furnished in permissible mesh. Enameled wire is very durable but a trifle too dense. It must be 14-mesh. For summer houses and sleeping porches and wherever the screened area is relatively large, it is most serviceable.

For tents, summer cottages and temporary abodes a processed waterproof mosquito netting is available. It is good for one season or several vacations.

Mosquito Bars.—A bar is no protection if the child can come in contact with it from the inside, or if it hangs loosely over the edge of the crib. The lower portion should be reinforced with sheeting, and the bottom tucked under the mattress all round.

Doors and Windows.—These should have weather strip closure all round. They should be very substantial to prevent sagging and warping. The writer has doors channeled all round on the edges and the groove filled with a bead carrying a double strip of thick ducking, projecting 1/2 in. This permits a loose fit in all kinds of weather. Frequently used openings should be vestibuled, the area between being 6 ft. deep. The doors must swing outward and never both ways. If a porch screen is over a stoop there must be a flush landing large enough to pass the open door going in and out.

Windows.—Check rail windows cannot be effectively screened unless the cloth is battened on the outside casing, which prevents washing the window. Any movable or adjustable screen to a window admits insects. Telescoping screens "to fit any window" should be prohibited by law. The proper window in mosquito territory is a casement, opening inward, with a fixed screen outside. A screen "bay" or screened porch outside the window are the only alternatives.

Other Openings.—Fire places, air ducts, wire and pipe passages, porch drains, inside cisterns and other nuisances too numerous to mention make effective screening well-nigh impossible. The chief causes of leaks are coarse mesh, defective wire, imperfect closure and propped-open doors.

Mosquitos in the house die in a week if deprived of water or moisture in any form. The vestibule should be rid of mosquito at frequent intervals. In the morning twilight the pests can be captured at the screen.

In cities, a well-directed campaign against breeding places is cheaper and more effective than the best screening.

Treatment of the Disease.—The use of the whole cinchona bark has been abandoned and not entirely with justice. The whole bark is relatively less toxic and more effective, but it is constipating and not very convenient for administration. The powder may be stirred with some syrup or jelly and given in a spoon. Quinin tannate is a very popular salt, as it is tasteless. Euquinin is just as palatable, more insoluble, and therapeutically more active.

The relative values of the quinin salts are as follows:

Salt	Per cent. alkaloid	Solubility in water
Quinin anhydride	100	1750
acetate	84	Slightly
bimuriate	71	Less than weight
bisulphate	59	8.5
citrate	67	820.0
hydrobromid	76	40.0
lactate	78	10.0
hydrochloride	81	18.0
salicylate	68	77.0
sulphate	74	720.0
tannate, about	30	800.0
valerianate	76	53.0
Euquinin	81	12500.00

(From Deaderick, modified from Laveran.)

To this may be added, cinchona bark, combined alkaloids, 15 per cent. Solubility, perhaps, 1:2000 for the alkaloids.

Experienced physicians in the South give a laxative before beginning quinin treatment, provided there is still time to prevent the next paroxysm. It always improves the well-being of the patient and makes him more tolerant of the quinin. Treatment should not be begun until the present paroxysm has passed the fastigium. Nothing can be gained by adding the discomfort of cinchonism to the travail of the fever. In the "benign" malarias during the agony of the fever a small dose of aspirin is permissible. The chill itself is relieved by a very small dose of morphine. These considerations for the comfort of the patient should not replace or postpone the specific treatment if there is urgency. The first dose of quinin can be given with the aspirin.

Dose and Mode of Administration.—In our clinics we prefer to give three large doses in 24 hours. Sometimes it is desirable to split these, doubling the number of doses. The maximum dose in any case is a total daily amount of ¼ gr. of alkaloid or its equivalent for every pound of body-weight. Half the quantity is sufficient in vivax or malariæ. The larger dose can do all that may be expected of quinin. The extraordinary doses sometimes given in intensely malarious districts are dangerous and uncalled for.

The whole bark should be used in cases of idiosyncrasy or intolerance. For very small children euquinin is the salt of choice. Larger children can swallow capsules of the more soluble salts, generally the sulphate. If the dose is followed by a hot lemonade, solution of the capsule and its contents is facilitated. It is never necessary to give quinin in solution. If the powder is given in wafer cachets and followed by an acid drink every requirement for dissolving quinin is met. During the administration of quinin the diet should be kept as low as possible, but as a rule, quinin should not be given on an entirely empty stomach as it is an irritant. The specific treatment should be continued in full dose for five or six days after the temperature has remained normal, and for 10 days longer in half doses. Then it should be given intermittently for a month or more, according to the duration and severity of the infection. It is true that this is not usually done, but if the blood is to be sterilized there is no escape from continued active cinchonization.

Of the three forms *vivax* is easiest to cure, *falciparum* comes next in order and *malariæ* last.

Other Modes of Administration.—In pediatric practice quinin often given by inunction and by suppository. We mention these only as methods not to be used; they are useless and unnecessary.* Only two other methods are available; the intramuscular and intravenous. Injections under the skin or into the fat are strictly contra-indicated. For local (intramuscular) injection the basic hydrochlorid may be used or the dihydrochlorid with or without urea. The last is painless if well diluted and slowly injected. The daily amount is ⅛ gr. per pound, given in two doses. For intravenous injection one can purchase sterile solution of the dihydrochlorid, 10 gr. in 10 c.c. The dose is 0.1 c.c. of the solution per pound of body-weight. It is safer to use in a dilution of 1:10. In young babies the jugular or posterior temporal veins are most conveniently selected for injection, or failing with these the median basilic vein may be exposed. Gold or platinum-iridium needles should be used.

Indications for Use by Needle.—The intravenous route is contra-indicated in case of idiosyncrasy and hemoglobinuria. It is the method of urgency, but quinin introduced directly into the circulation is rapidly eliminated. The method is very dangerous and not ordinarily justifiable. In the case of children, then, it should be reserved for cases of great urgency and would consequently not have to be repeated in any single case. The intramuscular route affords gradual absorption and must not be relied upon as a first dose in severe infections. It may be used when there is gastric irritability, urticaria, intolerance or idiosyncrasy and when doubt exists as to compliance with orders.

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* Suzuki, "Sur la quinothérapie dans le paludisme des enfants," Arch. d. Med. d. Enf., Tome xxv, i (1922), pp. 1–14. Shows by extensive experiment in the laboratory and upon patients that: The employment of the hydrochlorate of quinine in a dilution of 1 : 200 to 1 : 400 of saline, administered by rectum is the most efficacious method of treating malaria. In these dilutions there is no irritation and the solution is retained until absorbed.

From his case histories we note that he gives single injections of from 20–50 c.c. per year of age of the 1:400 dilution. In none of the four cases cited was there a recurrence.

Technique for Intramuscular Injection.—The patient should lie on his side with thighs flexed on the abdomen. The upper gluteal region should be thoroughly cleansed and iodin applied at the site selected for injection. Then, using a long needle, the sterile solution should be injected slowly well beneath the subcutaneous fat. The site of injection should be massaged for a short time.

Idiosyncrasy.—Absolute intolerance, involving dangerous symptoms, even to the bark or the combined unsalted alkaloids, is extremely rare. Desensitization has been successfully practiced.

Substitute for Quinin.—A very large experience in several hospitals justifies us in saying that effective substitutes do not exist. Bromides in some form or the whole bark instead of quinin will help us over any difficulty encountered. Among the agents tried we can enumerate salicin, salicylic acid, methylen blue, arsphenamin, the arylarsonates, tartar emetic, emetin, picric acid, mercuric chlorid, fluorescein, *x*-ray, ultraviolet light, etc. Acetanilid is toxic for the parasites but nearly as toxic for the patient.

Collateral Measures.—The cold pack in hyperpyrexia, cool spongings, an alcohol rub and warm fomentations during the chill and in algid malaria will suggest themselves to the trained physicians. Dyspnea and cardiac distress are treated according to physiological principles. In our hands saline enemeta or hypodermoclysis are preferred to strychnin and digitalis. Morphine and atropin restore vasomotor balance disturbed by chill, algidæ, syncope, etc. Gastric disturbance is often arrested by an emetic. Mild sinapisms, chopped ice or sherbets, carbonated limeade, hot lemonade, and, in extreme cases, novocain may be given in such cases. Adrenalin in drop doses has been recommended. Opiates hypodermically are the last resort. Food must be withheld until the gastric hyperemia subsides. During this time fluids may be given by proctoclysis. In comatose malaria with hyperpyrexia the right heart may be overloaded. Amyl nitrite, nitroglycerine of even venesection may be needed.

Provocative treatment has no place in pediatric practice.

It is customary to prescribe arsenic in convalescence. In all forms of toxic anemia and especially in malarial cachexia its use is believed to be indicated. So far as our experience goes, Fowler's solution, one tenth drop daily per pound, gradually increased to tolerance has been of considerable value. Organic arsenic should not be injected into the site of quinin indurations. Sodium cacodylate is very popular.

To Recapitulate.—Give a dose of castor oil or saline aperient or even small doses of calomel at once, if there is time. Let the first dose of quinin be a double one and then give the daily dose divided into three to five equal portions. In case of urgency the laxative or cathartic may be postponed, but not dispensed with. Keep the patient comfortable, treat complications, keep the diet low, and *sterilize your patient* before considering your responsibility at an end.

Relapses.—There is another hoary tradition concerning the periodicity of relapses. It probably had its origin in the old direction to repeat treatment every 7, 14 or 21 days. This form of direction is useful because a certain day of the week is set aside for repeating the dose. Just how a 48 hour parasite could relapse in seven days has not yet been satisfactorily explained. The literature shows relapses on any odd day up to months after infection. As stated under "Collateral Causes" some incident lowering resistance ushers in the chill. Relapses are frequent if thorough and intensive follow-up treatment is not instituted. Unfortunately, we have no means of differentiating relapses from reinfections.

Treatment of the Cachectic State.—If parasites are present they must be eradicated. Usually a general tonic regimen with rest and avoidance of exposure are the only indications.

Treatment of Gamete Carriers.—The opinion formerly held that microgametocytes are less resistent than macrogametes and disappear earlier is erroneous. This theory which has been defended principally by Ruge and Maurer, seems to have been disproved by Thomson, using Ross and Thomson's thick film counting method. There appears to be no recent proof that quinin has any effect upon gametes, but there is evidence that they disappear after persistent treatment for three to four weeks. Darling, Bass and Thomson have failed to find gametes after four weeks, and find them to diminish in number much earlier. It is believed that four weeks is perhaps the duration of life within the blood and that the quinin simply checks their production.

Special treatment for the control of gametes has been variously tried. Arsenic, arsphenamin, Roentgen-ray, mercuric chlorid, tartar emetic, all have had their advocates. Leroy reports having sterilized the blood by at most three intravenous injections of 1/4 gr. mercuric chlorid. We can say that no remedy has yet been found which has been shown to have any influence whatever upon gametes.

In the case of persistent relapses, grave anemia, dropsy, alarming cachectic state, and all other conditions indicating failure to sterilize the blood, change of climate is the only remedy. Many cases of malarial infection undergo spontaneous cure after frost.

Rest in bed and light diet often bring about a spontaneous clinical cure. No spontaneous cure can be relied upon, except that following a change of climate, and even then remote relapses have occurred.

ADDENDA

The foregoing Mss. was completed before any of the extremely voluminous literature on War experiences with malaria was available. Although the experience with debilitated soldiers under the stress of active service finds no direct application in the treatment of children, some of the findings are of great interest.

I. Weselko, O., Congenital malaria., Zeitsch. f. Aertztl. Fortb., xix (1922), pp. 432–433 reports: "Of 200 newborn of malarial mothers 187 were found to have estivo-autumnal parasites in the blood. Of these 144 died in the first week and 28 in the second week."

One wonders how many such women must have aborted.

II. Treatment and Relapses.—The National Malaria Committee in 1919 reported favorably on the treatment adopted by Bass in certain counties in Mississippi during 1917: "For the acute attack, the adult dose is 30 gr. daily of the sulphate by mouth, divided into three doses. For the sterilizing of the patient, 10 gr. of the salt are given by mouth in a single dose every night for eight weeks." The proportionate doses for children are given in detail. This treatment was supposed to "sterilize" 90 per cent. of persons treated. This is referred to abroad as the American Standard Treatment.

In all the literature great disappointment is expressed as to the inadequacy of quinin in the definite cure of malaria. Col. Sir Clayton Lane, in a splendid summary of the war experiences in the treatment of malaria, says: "The main curative interest continues to rest with quinin despite numerous disappointments . . . we are gradually reaching a better appreciation of the limitations of the drug and thus of its value."

The consensus of opinion is reflected in the statement by Frazer: "The disheartening thing to all patients is the recurrence of relapses in spite of quinin. Any faith they have ever had is severely shaken. It is still the best drug."

Noteworthy are the reports of Acton, Rennie, Curjel and Dewey. Soldiers heavily infected in the Macedonian campaigns were treated in Dagshai, in the sub-Himalayas where endemic malaria is unknown, and observation upon relapses must be deemed reliable. Contrary to existing belief, estivo-autumnal malaria was found to be more curable than tertian. No observations upon quartan malaria were made.

Of 663 cases of tertian malaria treated in six groups by different anti-relapse methods, the average cure percentage from a single course of treatment was 32.4, the lowest being 18 per cent., the highest, 52 per cent. The 18 per cent. group comprised 100 patients, who received two to five intravenous injections, followed in most cases by oral quinin for eight weeks. The 52 per cent. group received, orally, 30 gr. daily for 21 days and 15 gr. daily for 90 days, making a total of 1980 gr.

Of 131 cases of estivo-autumnal fever, only 13 cases relapses (Tropical Diseases Bulletin, xvii, p. 5).

Recent reports by authors in different countries can be summarized as follows:

Ross type of treatment. Continuous intensive oral administration for more than five weeks, 30 to 70 per cent. relapses.

Bass Type. Continuous treatment in reduced doses after clinical cure (no relapse data).

Stephens. (The best of 10 methods tried.) Full doses followed by week-end full doses, not less than eight weeks (22 per cent. relapses).

Nocht. One gram in five doses daily for 10 days and then two days together with gradually lengthening intervals (relapses "few").

Rawnsley (Jl. Royal Army Medical Corps, xxxi (1918), pp. 60–75 and 272–276). Ten grains daily for any length of time failed to prevent relapses.

The proportion of relapses to primary cases in the post-war literature, including both civil and military practice, gives figures varying from 36 to 90 per cent.

From a large group we select Bass, as representing average conditions in American homes. He says, "In areas of great prevalence, from 50.77 to 68.86 per cent. of all attacks in a given year are relapses" (Eighth Ann. Report, Int. Health Board (1922).

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CHAPTER CXXXV

INFECTION AND IMMUNITY

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INFECTION

By infection is meant the invasion of the living tissues of one species by individuals of another species which multiply and functionate at the expense, and to the detriment, of the host. The invading organisms may be an ultramicroscopic virus, as in pleuropneumonia and foot and mouth disease of cattle; or bacteria, as in typhoid fever and pneumonia; or protozoa, as in malaria and sleeping sickness; or metazoa, as in uncinariasis.

The ability of microörganisms to produce disease depends, therefore, upon their faculty of entering the body of the host, of finding suitable nutrition therein, and of multiplying in the tissues and body fluids. By far the greatest number of known species of bacteria do not possess this power. They are unable to invade the living animal body, or having entered it accidentally, are unable to survive. These are *saprophytes*. They are the agencies which carry out that sector of the nitrogen cycle in which the complex organic compounds which compose plants and animals and their products are degraded to simpler mineral substances. Their most distinctive quality is their powerful chemical activity.

A much less numerous group of bacteria are *parasitic*, that is, they exist upon or in the living bodies of animals. Kendall* has pointed out that their chemical activity is much less vigorous than that of saprophytic bacteria, and more nearly approaches that of the cells of their animal hosts. From these parasitic bacteria, a smaller and more formidable group of microörganisms has developed, namely, the *pathogenic*. Kendall* has distinguished certain differences between the disease producing powers of these two groups. Parasitic bacteria, such as B. coli and staphylococci, are found upon the skin and mucous membranes and in the respiratory and gastro-intestinal tracts of humans; they are "opportunists" and only invade the body when some special occasion offers; and the diseases produced by them are non-specific in character and almost never epidemic in distribution. Pathogenic bacteria, on the other hand, like B. tuberculosis, B. typhosus, etc., readily invade the bodies of their hosts; the diseases which they induce are specific; and, because of their perfected mechanism of entry into and exit from the host, they may cause widespread epidemics of disease.

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* Kendall, A. I., Boston Med. & Surg. Jour., clxix (1913), p. 749.

Infection may be thought of as a combat between the invading microörganism and the invaded animal host. This conflict may pass through all of the vicissitudes of any battle from a quick defeat for one or the other of the combatants to a permanent stalemate. From the standpoint of the infecting microörganism, the last condition, a sort of "peace without victory," is the most desirable. Infection may also be looked upon as a biologic process with some of the characteristics of a struggle for existence. The end to be attained by the invading microbe, however, is not the replacement of one species by itself, but the bringing about of such a balancing of forces as will enable the two to live together comfortably.

Theobald Smith* has pointed out the essential elements in the life history of any pathogenic microörganism. These are the necessities (1) of finding a portal of entry into the body of a host; (2) of securing nutrition and multiplying therein; and (3) of finding a portal of exit from the body of one host and the means of reaching new hosts. A microbe of too great virulence may destroy its host before it is able to escape to another, and its family history comes to an end. Those microörganisms which cause such specific and characteristic diseases as tuberculosis, typhoid fever, malaria, plague, and gonorrhea, have perfected all of the phases of this cycle of pathogenism.

As suggested by Doctor Welch† in his Huxley Lecture, pathogenic bacteria possess means of defense against the host which they invade. There is perhaps no disease-producing microörganism that has offensive weapons as such. The toxic substances by which the infected animal is killed are either normal products of bacterial metabolism, such as the soluble toxins of the diphtheria bacillus; or they are an integral part of the body protein of the microbe, such as the endotoxins of the typhoid bacillus. In either instance, the substance which proves so deleterious to the animal host is produced by the microörganisms without regard to whether they are growing in such a host or in an artificial medium. They are a part, therefore, of the fundamental life processes of the bacteria, and are no more to be considered as weapons of offense than are the digestive juices in higher animals.

The means of defense possessed by microbes are probably among the latest characteristics to be acquired in the process of their evolution, and are among the first to be lost when a microörganism, accustomed to subsisting upon a living animal, is forced again to take up a saprophytic existence. Upon the perfection and efficiency of this defensive mechanism, the virulence of bacteria largely depends.

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* Smith, Th., Amer. Med., viii (1904), p. 711.

† Welch, W. H., Johns Hopkins Hosp. Bull., xiii (1902), p. 285.

FACTORS DETERMINING THE CHARACTER AND INTENSITY OF INFECTION

The character and intensity of an infection depend upon (1) the species and virulence of the infecting microörganism; (2) upon what may be termed the "physiologic ensemble" of the host; and (3) upon external factors that may be considered under the general term, environment, which affects both the microörganism and the host.

1. Factors Pertaining to the Microörganism.—Infections by some microbes are acute, the patient either succumbing quickly, as in bubonic plague or Asiatic cholera; or recovering promptly, as in measles and dengue fever. Other infections are characteristically chronic, as leprosy, tuberculosis and syphilis. The lesions produced by some species of bacteria are sufficiently characteristic to justify a probable diagnosis of the infecting microbe from the anatomic changes observed in the host. This is especially true of the lesions due to tubercle bacilli; the different stages of hepatization of the lung induced by pneumococci; and the ulcers of the intestine in infections with typhoid bacilli. Other varieties of microörganisms are distinguished by the diverse character of the lesions produced. Perhaps the most versatile of bacteria is the streptococcus which produces such multifarious pathologic processes as erysipelas, cellulitis, septicemia, septic endometritis, bronchopneumonia, and septic sore throat with its train of disastrous complications.

The species of bacteria also determines whether the resulting infection will be marked by toxemia or bacteremia. Thus B. diphtheriæ and B. tetani remain localized and their toxins, being absorbed, cause the symptoms observed clinically. B. typhosus, on the other hand, is found in the circulating blood in the earlier stages of the infection. Different varieties of bacteria also show a preference for certain organs. The meningococcus rarely produces lesions elsewhere than in the meninges; the typhoid bacillus shows a predilection for the lymphadenoid tissue within the abdominal cavity.

Different types or varieties of the same species of microörganisms show differences in the character and intensity of the infection which they cause. In many instances these types cannot be differentiated by cultural means but only by serologic tests. Cole, by means of specific agglutinins, has differentiated four groups of pneumococci which exhibit certain differences in the type and severity of infection. The great differences between the pathogenic powers of the human and bovine types of the tubercle bacillus for rabbits is well known. The lesions produced in humans by these two varieties of B. tuberculosis also show characteristic differences, those of the bovine type being usually more massive and tumor-like than the lesions of the human type.

In order to produce infection it is necessary that at least a certain minimum number of microörganisms enter the body at one time. This is because of the large number of casualties among the first invaders before they can adapt themselves to their new environment in the face of the active resistance offered by the invaded host. This minimum number varies with the species of microbe, with the degree of virulence possessed by different strains of the same species, and with the powers of resistance of the invaded host. A single plague bacillus and a single thread of anthrax bacilli have produced infection in the guinea pig and the white rat, respectively. Webb and Williams found that 20 tubercle bacilli injected subcutaneously into a guinea pig produced tuberculosis in the animal. In order to induce pneumonia in a dog it is necessary to introduce massive doses of pneumococci deeply into the bronchi.

The place of entry of a microbe into the body determines to a large extent the specific nature of the disease which it produces. The strictly pathogenic microörganisms are those which have so perfected their cycle of pathogenism that they enter and leave the body of their hosts in a fairly definite and constant manner. The "opportunist" parasites have an imperfectly developed cycle, and their entrance and exit, as well as the results of infection, are inconstant. The same species of microbe entering the body by different routes may cause different types of infection. When B. pestis penetrates the skin it produces the common form of bubonic plague; when it enters the body through the respiratory tract, it leads to a foudroyant infection of the lungs, pneumonic plague. B. anthracis may produce a malignant pustule; or a violent inflammation of the trachea, bronchi, lungs and regional lymph-nodes (woolsorters' disease); or a hemorrhagic enteritis (mycosis intestinalis); according to whether it enters the body by way of the skin, the air passages or the gastro-intestinal tract.

Virulence is the power of a microörganism to produce pathologic change after it invades the animal body. Once it has gained entrance into the tissues and body fluids, its ability to maintain itself there will depend upon its powers of passive resistance and of active defense, rather than upon any actual means of aggression which it possesses. The formation of capsules by bacteria is a means of passive resistance. The virulence of pneumococcus, B. welchii, and B. anthracis increases with the readiness with which they form capsules; while the virulence of the habitually capsulated microörganisms, like B. mucosus capsulatus, and streptococcus mucosus, is reasonably constant. The clubs produced by actinomyces when growing in the body form a compact phalanx which probably protects the microbe against the invasion of the leukocytes that crowd about the mass.

Many bacteria, however, possess means of defense of a more active nature. Thus B. welchii and other microörganisms produce negatively chemotactic substances which repel phagocytes from the infected regions. In the case of B. welchii this may be due to the lactic acid produced by the fermentation of the glycogen of the infected muscle. Under the term "aggressins," Bail* has described more or less theoretical substances which, he believes, bacteria produce under the stress of a struggle against the unusual defenses which they meet in the animal body, and upon which their virulence depends. Whatever the nature of aggressins, whether they are merely extracts of dead bacteria (endotoxins), as claimed by Wassermann and Citron, or are actual secretory products of the bacteria, their action is defensive rather than offensive. The virulins described by Rosenow* in cultures of virulent pneumococci are likewise purely defensive substances. Their presence prevents phagocytosis, not by injury to the leukocytes, but by neutralizing the effects of opsonins upon the microbes. The marked variations in virulence of different species of bacteria, and of different strains of the same species, depend upon the degree of efficiency of their mechanisms of defense.

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* Bail, Arch. f. Hygiene, lii (1905), p. 342.

The virulence of a given strain of bacteria may be modified by several means. It may be either increased or decreased. The growth of bacteria upon artificial media even for short periods of time almost always results in diminution of their pathogenic properties. Different species of bacteria show great variation in the readiness with which they become non-pathogenic. Staphylococcus aureus rather promptly loses its virulence. B. diphtheriæ, on the other hand, is very retentive of its toxin-producing power. This difference is probably due to the fact that toxin-production is a normal metabolic activity of the diphtheria bacillus, and is essential to the life of the microörganism whether it is growing on a mucous membrane and producing disease, or in an artificial medium. The means by which the staphylococcus maintains itself in the tissues and body fluids, that is, the mechanism of its virulence, are temporary attainments that are readily lost when the environment is changed.

Kendall† and his co-workers have shown that the fundamental metabolism of most pathogenic bacteria can be greatly modified by the addition of a fermentable carbohydrate to the culture medium. This change in metabolism is probably associated with an alteration in virulence.

Pasteur observed that growing anthrax bacilli at a temperature a few degrees higher than that of the human body resulted in a lowering of its virulence. He employed this method to secure cultures for immunizing sheep against the disease of anthrax. Other varieties of bacteria are similarly affected by the same means. The presence of small amounts of chemical germicides, like phenol, in the culture medium also induces a reduction of virulence. Most bacteria also suffer a diminution or complete loss of virulence as a result of drying.

The habituation of a given variety of bacteria to growth in the bodies of one species of animal usually lowers it virulence for other animals. The bovine type of tubercle bacillus is only slightly pathogenic for man. The "fixed virus" of rabies, so powerfully virulent for rabbits, is said to be almost non-virulent for man.

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* Rosenow, E. C., Jour. Infect. Dis., iv (1907), p. 285.

† Kendall and Farmer, J. Biol. Chem., xii, xiii (1912); Kendall, Day and Walker, J. Amer. Chem. Soc., xxxv (1913), pp. 1201–1249.

There are fewer methods of heightening the virulence of microörganisms at our disposal than for lowering it. Growing bacteria upon media containing unheated, that is, uncoagulated, body fluids, such as ascites and hydrocele fluids, blood and blood serum, does not so much increase their virulence as it prevents the loss of it. The surest method of increasing virulence of a given variety of microbe is that used by Pasteur in producing the "fixed virus" of rabies, and known as "animal passage." By inoculating a rabbit subdurally with an emulsion of the brain of a dog dead of street rabies, it was found that the animal died quite regularly at the end of about 21 days. By subdural inoculation of a second rabbit with an emulsion of the brain of the first, a third with the brain of the second, and so on through a long series of rabbits, there was evidence of a steady increase of virulence in the reduction of the time during which the animal survived after inoculation. This period could not be reduced below seven days, however. When this point was reached, this potent virus was called "fixed virus."

2. Factors Pertaining to the Host.—Microörganisms which are highly virulent for one species of animal may be wholly non-pathogenic for another. The filterable virus of pleuropneumonia of cattle, the bacillus of swine plague, and the bacillus of chicken cholera, while producing fatal infections in these species, are non-virulent for man. The mechanism of this phenomenon may have a different basis in different instances. The factors concerned in some cases may be purely physical, in others they may be more subtle, and difficult to discover.

The normal body temperature of different animals varies greatly, from that of cold-blooded animals whose temperature is approximately that of the surrounding medium, to that of birds which ranges from 104 to 106°F. Pasteur was at first unable to produce infections in chickens with anthrax bacilli. But by standing the fowls in water until their temperature was reduced, they developed the disease. The optimum temperature for the growth of the avian type of tubercle bacilli is several degrees above that of the human body. Birds are not readily susceptible to infection with the human type, nor are men and cattle easily infected with the avian type.

Just as different animals employ different methods of defense against visible enemies, so also the means of combatting infection will vary, more or less, with the species of animal concerned. Thus the dog and the rat are relatively resistant to anthrax. The resistance of the dog is dependent upon phagocytosis; that of the rat upon bactericidal substances in its blood.* A microörganism that is accustomed to meeting the defensive and offensive mechanism of one animal host may be altogether incapable of protecting itself against that of another

* Hektoen, L., Jour. Am. Med. Assoc., xlvi (1906), p. 1407.

species. The serum of the rabbit contains a powerful lysin for typhoid bacilli. When any reasonable number of these bacilli are injected into a rabbit they are quickly destroyed. The pneumococcus, on the other hand, is able to protect itself, perhaps by means of its capsule, against any lysin that might destroy it, and by means of "virulin" against phagocytosis. This microbe, therefore, multiplies rapidly in rabbit's blood and causes a fatal septicemia.

It is usually difficult and frequently impossible to produce in animals, by the injection of microörganisms pathogenic for man, the type of lesion characteristic of that infection in the human species. It is difficult, for example, to produce a typical pneumonia in rabbits by the intratracheal injection of pneumococci. These animals usually die of septicemia before a pneumonia has time to develop The lesions which are so characteristic as to be pathognomonic of an infection with a given microbe in a host to which it has become adapted, are the result of the dynamic balance between the defensive mechanism of the invaders and the offensive and defensive tactics of the invaded host. A difference in any element of either of these mechanisms will lead to deviations from the usual type of lesions induced by a given microörganism.

Furthermore, the body fluids from which the invading microörganism must obtain its nutriment, differ chemically in different animals. It is for the reason of this double adaptation to food requirements and defense mechanism, that it is necessary to use special animals for the artificial production of certain diseases. Thus such humanized diseases as typhoid fever, poliomyelitis and syphilis can only be produced in anything like their typical pathognomonic form in animals that are closely related generically to man, as the monkeys and the anthrapoid apes.

Different varieties of the same species of animals frequently show quite unexplainable differences in susceptibility to infection with the same microörganism. Chauveau found that Algerian sheep resisted a dose of anthrax bacilli to which French sheep regularly succumbed. Jersey cattle are very susceptible to tuberculosis; the Holstein breed is relatively resistant.

It was formerly thought that the crowding of children into ill-ventilated schoolrooms was not the only factor concerned in the marked predominance of scarlet fever, measles, diphtheria, whooping-cough and chicken-pox in that period known as the "school age;" but that there was reason for believing that young children are actually less resistant to these infections than are adults. The experience of the American Expeditionary Forces in France has cast serious doubt upon this former belief. Neal and Sutton* found that mumps, measles, scarlet fever, and diphtheria, in the order named, ranked as the most important causes of days lost in hospitals by soldiers. The data which they present indicate that opportunity for exposure due to overcrowding in barracks was the chief, if not the sole, cause of the high morbidity of these diseases among the American troops, and that adults are quite susceptible to the so-called "diseases of childhood."

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* Neal and Sutton, Military Surg , xlv (1919), p. 421.

Age influences the type and severity of certain infections. The severer forms of tuberculosis in children are generally of the bones and lymph-nodes; in adults, of the lungs. Lobar pneumonia in children is rarely seen at the autopsy table, bronchopneumonia being by far the more common type encountered. Brückner* concluded from his epidemiologic studies in Treves, that typhoid fever is a very common disease in children, and that in them it is usually so mild and of such short duration that it usually goes unrecognized. Such children may be sources of serious danger in the spread of the disease.

Every epidemic of disease reveals numerous instances of individual differences in resistance to infection. Until recently there was no reliable means of distinguishing the susceptible from the non-susceptible. The intracutaneous injection of a minute quantity of diphtheria toxin, known as the Schick test, is a method of distinguishing these two classes with reasonable certainty so far as diphtheria is concerned. The appearance within 24 hours at the site of injection of an area of redness surrounded by a diffuse zone of induration constitutes a positive reaction and indicates the absence of sufficient antitoxin present in the blood to neutralize the poison injected. A negative Schick test indicates a degree of resistance sufficient to warrant the withholding of an immunizing dose of antitoxin in persons who have been exposed to infection with diphtheria. It is not improbable that a low individual resistance to one type of infection may be associated with a similar condition for other diseases. This test may, therefore, be found to furnish an index to the general susceptibility of a patient to infection. Zingher† found that the incidence of measles, scarlet fever, and poliomyelitis was much greater among children who had given a positive Schick test than among those who reacted negatively.

When an infectious disease makes its appearance for the first time in any population, epidemics of it are much more wide-spread and are accompanied by a greater mortality than among peoples accustomed, through many generations, to such an infection. An extremely high death-rate followed the introduction of measles among the American Indians and among the natives of New Zealand. In 1873 malaria was introduced into Madagascar and literally decimated the population of that island.

On the other hand, many diseases are notoriously milder in those regions in which they are endemic, than they are in countries where they occur as occasional epidemics. Yellow fever shows a lower mortality on the east coast of Mexico than it does during the occasional epidemics in the United States. Again, many infectious diseases have shown a tendency to become milder after existing for many generations among a given population. There is evidence that syphilis is a much less severe and acute infection in Europe at the present time than when it first made its appearance there in the fifteenth century.

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* Brückner, Münch. med. Wchnschr., No. 23 (June 7, 1910).

† Zingher, Am. J. Dis. of Children, xiii (1917), p. 247.

This difference in the character of infectious diseases in a recently and anciently infected population may be due in part to an immunity developed as a result of unrecognized mild infections in early life and in part to an inherited mechanism for combatting the infection. In a people infected for the first time, with any given microörganism, the defensive and offensive mechanism of the body are not adapted to successfully contending with such an infection, and the incidence of the disease is large and the mortality is high.

Tuberculosis has been styled a "class disease" because of its high morbidity and mortality among those who suffer from the debilitating effects of under-nourishment, bad sanitary conditions, and excessive fatigue. Not only are the opposite conditions of life, and the finer physique that usually goes with them, an effectual protection against this disease, but the transference of a patient in the earlier stages of tuberculosis to an environment in which he can obtain abundant food, fresh air and rest frequently results either in an arrest of the disease or in a "permanent cure." Fine physical vigor is little or no protection, however, against typhoid fever, pneumonia, small-pox and similar diseases. With equal opportunities for infection, these diseases attack the strong and well-nourished as readily and as fatally as they do the weak and emaciated.

Certain diseases, ordinarily mild in themselves, predispose to more serious infections. Thus uncomplicated measles and influenza are rarely fatal; but the pneumonia which so frequently complicates them accomplishes a frightful mortality. Diabetics appear to be especially susceptible to furunculosis and tuberculosis. In many other chronic diseases the chapter is closed by a "terminal infection," like pneumonia or septicemia. The writer has observed at autopsy several instances of very violent and rapidly fatal infections in persons of the lymphatic constitution.

3. Factors Pertaining to the Environment.—In passing from one host to another, pathogenic bacteria are frequently subjected to irregularities of temperature, to uncertainties in food supply, and to competition with saprophytes, for variable periods of time. This usually results in the reduction or loss of virulence, and perhaps of other recently acquired characteristics. The effects of light and drying are also deleterious to the microörganisms. The free oxygen of the air quickly destroys pathogenic anaërobes which do not promptly form spores.

In the case of protozoan parasites, environment may result in lack of opportunity for passage to new hosts, if climatic conditions are such that the intermediate insect host cannot exist, or if these insect hosts are excluded by proper screening.

The effect of environment upon the human host is one of opportunity for infection or the lowering of individual resistance.

Sources and Modes of Infection

Infection may be transmitted through the air; by means of food and water; by animals; by direct or indirect contact; and by way of the placenta before birth.

Air-borne Infection.—Bacteria which adhere to minute solid particles of dust that float in the air may be subjected to two very injurious influences, drying and sunlight. Drying tends to decrease virulence, and if sufficiently prolonged causes the death of the microörganisms. The resistance of different bacteria to drying varies greatly. Thus the gonococcus is destroyed in a very few hours; diphtheria bacilli after 20 to 30 days; and tubercle bacilli after two to three months. Bacteria subjected to direct sunlight are quickly killed. In diffuse light they succumb only after a longer time. Thus Koch found that tubercle bacilli were killed by direct sunlight in about two hours, but survived for several hours in diffuse daylight, and for a much longer time in dark places. Hence the dust of darkened rooms is much more likely to convey infection than that encountered out-of-doors.

In the acts of coughing, sneezing and talking, fine particles of spray are emitted from the mouth and nose. The larger of these settle rapidly to the floor, but the extremely minute particles may float in the air for many hours. In violent coughing they may be driven out many feet in front of the face. In open tuberculosis, in diphtheria, in epidemic meningitis and probably in scarlet fever and measles, the saliva and nasal secretions may contain the microörganisms of the disease, which are carried out in the spray caused by coughing and sneezing. By the inhalation of these minute particles or droplets a new host takes into his upper air passages microörganisms that have not been subjected to the deleterious effects of sunlight and drying and their virulence is not reduced. Obviously, droplet infection is a most important method of transmitting infectious diseases of the respiratory tract.

Water and Food as Sources of Infection.—Diseases of the gastro-intestinal tract are usually, and of the respiratory tract sometimes, transmitted by water and food. Water becomes contaminated either directly or indirectly by being mixed with sewage or human excreta. Public water supplies may become contaminated by sewage emptied directly into the source from which the water is obtained, or from excreta carelessly disposed of on the slopes of the drainage basin of the water supply. Pathogenic bacteria which get into such a water supply are subjected to certain destructive agencies, so that water thus polluted may later become harmless.

The diseases which are most commonly spread by means of water are typhoid fever, cholera and the dysenteries. Purification of public water supplies by proper sand filtration causes a rapid and marked reduction in the number of deaths from these diseases.

Food may become contaminated by the soiled fingers of persons handling it; by being kept in vessels rinsed with polluted water; by means of dust and droplets if left exposed; and by insects that mechanically carry infectious material. Certain foods are more dangerous sources of infection than others. Milk is especially liable to become contaminated. Its opacity renders detection of its contamination difficult, and it is an excellent medium for the growth of bacteria. Hence a moderate number of pathogenic microbes may, in a few hours, increase to very great numbers. Vegetables which are eaten raw, may be grown in land fertilized with "night soil" and thus become a source of danger. Oysters "fattened" in polluted river water may carry typhoid infection. Foods may also contain the poisonous products of bacteria which have grown in it; for example, the relatively thermostable endotoxin of Bacillus paratyphosus, and the more thermolabile, soluble toxin of Bacillus botulinus.

Soil.—Certain spore-forming bacteria, like the bacilli of tetanus, gas gangrene and anthrax, have their natural habitat in the soil and commonly enter the body through wounds of the skin. The soil is also the source of infection in hook worm disease.

Animals.—Many diseases of man are acquired through the medium of various species of animals. The transmission of infection may be direct, from an animal actually suffering from the disease, as in glanders and hydrophobia. Or, the animals may mechanically carry the infectious material upon or in their bodies. Flies may become contaminated with typhoid bacilli which are deposited on food upon which they alight or into which they fall. Blood sucking insects may carry in their bodies pathogenic microbes which they have taken in with the blood of infected persons bitten by them. These microörganisms may be re-injected when they bite a new host. Typhus fever and bubonic plague are thus transmitted by the louse and the rat flea, respectively. Plague bacilli may also be deposited upon the skin in the dejecta of the flea and rubbed in by the patient in scratching. In other diseases animals act as the intermediary host in which the parasite undergoes one of its life cycles. The Anopheles mosquito in this manner transmits the plasmodium of malaria; the pig, trachina spiralis; and fish and cattle, the tape worms, Dibothriocephalus latus and Tenia mediocanellata, respectively.

Contact.—Many infections are transmitted by direct or indirect contact with persons suffering from the disease or with those who have become "infection carriers." The venereal diseases are the most typical examples of infection by direct contact. But diphtheria, whooping-cough, scarlet fever and tuberculosis may also be acquired through kissing, exchanging gum and from common drinking cups.

It was found impossible to control epidemics of influenza, diphtheria, pneumonia and other infectious diseases in the American Expeditionary Forces until the soldiers were compelled to wash their mess kits in water that was boiling at the time the untensils were being cleansed.*

Prenatal Infection.—Germinal infection, that is, infection actually transmitted in the sperm or ovum, is questionable, and with the possible exception of syphilis, probably does not occur. Prenatal infection by way of the placenta, while relatively rare, does occur. Congenital syphilis is the disease most commonly conveyed in this way. Small-pox, measles, pyogenic infections, typhoid fever, and tuberculosis have also been thus transmitted to the fetus. Many obstetricians have become convinced that trans-placental infection is a common cause of stillbirths.

Portals of Entry and Exit of Infecting Microbes. Means of Defense against Infection

Great numbers of bacteria are constantly present on the skin. Of these, staphylococcus aureus and albus and streptococcus are, perhaps, the most important. The normal intact skin is a natural and effective barrier against the penetration of these microbes. Mechanical injury of the skin permits the invasion of the "opportunist" pathogens which cause infection unless they are quickly destroyed by bactericidal substances in the body fluids or by phagocytosis. Entrance is sometimes effected by way of a hair follicle, especially when aided by rubbing, as by a collar on the back of the neck. The majority of the infections that occur by way of the skin are non-specific inflammations like furuncles and carbuncles. When bacteria, especially streptococci, penetrate still more deeply into the tissues, they lead to cellulitis, a rapidly spreading infection of the loose connective tissue between the muscles; or to septicemia. Occasionally more specific infections occur through the skin, such as bubonic plague, tetanus, blastomycosis and hydrophobia. Other specific infections are closely related to occupation, as, for example, the so-called anatomical tubercles on the hands of butchers and morgue men, the malignant pustule of anthrax, and hook worm infection. Bacteria which have entered the body through the skin escape from the body again either in discharges through spontaneously or artificially made openings into the site of infection; or, if they penetrate more deeply and enter the blood stream, they may be excreted through the liver and kidneys.

Intact mucous membranes, like the intact skin, are natural and effective obstacles to invasion by the numerous bacteria that are present in the gastro-intestinal and upper respiratory tracts. The common bacterial inhabitants of the mucous membranes of the mouth, nose and naso-pharynx, are streptococci, staphylococci, and pneumococci of a type which Cole found to differ serologically from those commonly found in pneumonia. Occasionally diphtheria bacilli, meningococci, and the virus of poliomyelitis may be found on these mucous membranes of healthy individuals. Microörganisms of all kinds find their way into the upper respiratory tract but are, for the most part, mechanically prevented from reaching the lungs by the vibrissæ in the front part of the nose, by the moist secretions on the nasal mucous membrane, and by the cilia that crown the epithelium of the trachea and keep up a constant upward sweeping motion.

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* Neal, M. P., Major, M. C., U. S. A., Personal Communication.

The more common and important infectious diseases whose agents enter the upper respiratory tract are the non-specific serous or seropurulent rhinitis; and the specific diseases, diphtheria, influenza, pneumonia, tuberculosis and blastomycosis of the lungs, epidemic meningitis, and probably poliomyelitis and many of the exanthemata. The microörganisms of these diseases are discharged from the body chiefly in the secretions of the mouth and naso-pharynx as a fine spray emitted in coughing, sneezing and loud speaking. Direct contact and air-borne droplet infection are, therefore, the most important factors in the spread of these diseases.

Through the mouth, bacteria in extraordinary number and variety enter the body. The majority of these are harmless saprophytes, but with them there may enter, on occasion, microörganisms capable of causing progressive disease. In the stomach these microbes encounter the hydrochloric acid of the gastric juice which destroys not only the non-spore-forming saprophytes, but also the "opportunist" parasites swallowed with the secretions of the nasopharynx, and such pure pathogens as tubercle, typhoid, paratyphoid and dysentery bacilli; cholera vibrios and pathogenic amebæ. If, however, on account of some digestive disturbance, the gastric juice contains little or no free acid, pathogenic bacteria may escape destruction and pass through the pylorus into the intestine. Polluted water probably passes through the stomach too quickly for destruction of the contaminating microbes.

In the intestines of nursing infants, acidogenic bacteria, like Bacillus acidophilus and Bacillus bifidus, are the predominating bacteria, with smaller numbers of other bacteria like Bacillus coli.* In the adult intestine, bacteria increase in numbers from the duodenum to the region of the ileocecal valve where the living bacterial flora is most exuberant. Of these, Bacillus coli predominates, and with it are associated other potentially pathogenic bacteria of the "opportunist" type. Hence, appendicitis and the peritonitis which follows the escape of intestinal contents into the abdominal cavity from any cause, are infections of a non-specific inflammatory nature.

On the other hand, typhoid fever, amebic and bacillary dysentery and cholera are specific diseases whose causative microörganisms have reached the intestine after escaping the destructive action of the acid gastric juice. The infections which they induce are marked by a train of clinical symptoms that are more or less typical, and are characterized by pathologic changes which are, at least in typhoid fever and amebic dysentery, striking and distinctive. The causative agents of these diseases escape from the body in the dejecta, and reach new hosts chiefly by means of food and water polluted with them.

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* Kendall, A. I., "Bacteriology, General, Pathological and Intestinal," Chap. 30., 1st ed., Phila. (1916).

The secretions of the vagina are normally acid in reaction due to the presence of Döderlein's bacillus. This prevents the survival of pathogenic bacteria which gain entrance. If, however, an alkaline discharge from the cervix of the uterus neutralizes the acid of the vaginal secretion, pathogenic bacteria are able to survive and multiply. The common diseases of the male and female genital organs—syphilis, gonorrhea and chancroid—are due to microörganisms that have perfected their cycle of pathogenism. They are transmitted almost solely by direct contact.

INCUBATION PERIOD

The time which elapses between the entrance of pathogenic microörganisms into the body and the appearance of definite subjective and objective signs of disease, is spoken of as the incubation period. During this time, the microörganisms have been increasing in numbers, and the defensive powers of the host have been augmenting in strength. The process of infection and the manifestations of it in the host are the net result of the combined action of the defensive mechanism of the microbes and of the offensive and defensive mechanisms of the host. In typhoid fever, the bacilli are present in the circulating blood in the early stages; as the antibodies increase in quantity, the microörganisms in the blood diminish and finally disappear, at a time when the symptoms are most severe.

This and other facts have led Vaughan* to formulate a conception of the stages of incubation and of active symptomatology on the basis of the source of the protein destruction which takes place in the body. During the incubation period, the microörganisms are breaking up and utilizing for their own nutrition, the proteins of the host. With the multiplication of the bacilli, there comes an increasing production of bactericidal substances with gradually increasing destruction of the invading microörganisms and liberation of bacterial proteid, either as such or in the form of its split products. Hence, according to Vaughan's conception, the incubation period is the lapse of time between the entrance of the microörganisms into the body of the host, and the formation of sufficient anti-bodies to liberate bacterial protein in quantities capable of causing symptoms. Somewhat similar is the view of von Pirquet and Schick,† that the incubation period represents the time during which the infected host is becoming "allergic" or hypersensitive to the proteins of the infecting microörganisms.

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* Vaughan, V. C., "Proteid Split Products in Relation to Immunity and Disease," Phila. (1913).

† v. Pirquet and Schick, Wien. med. Wchnschr., xvi (1903), pp. 758 and 1244.

The length of the incubation period is determined primarily by the type of microörganism causing the infection, and within rather narrow limits the common infectious diseases have incubation periods that are uniform and characteristic. The actual duration of this period in any given disease is dependent upon the number and virulence of the microörganisms introduced, the powers of resistance of the host, and, in hydrophobia, upon the location of the wound that furnishes the portal of entry for the virus. In general, the shorter the period of incubation, the more severe and dangerous the infection. This is especially true of infections with streptococcus and Bacillus tetani.

How Bacteria Produce Disease. Local and General Reactions to Infection

The primary effect of the entrance of most pathogenic microörganisms into the animal body is a local irritation at the site of invasion. The further progress of the infection; the character of the lesions produced; the acuteness of chronicity of the disease; the manner, rate and extent of the spread of the infectious agents from the primary focus; and the final effect upon the host; are functions of many variables. The most important of these are the number, virulence and type of the invading microörganisms; the portal of entry to the body; and the resisting powers of the host. With a few exceptions, the first effect of the entrance of microbes into the body is a reaction on the part of the tissues about the site of the invasion, called inflammation. If the invading microörganisms are of the group of "opportunist" pathogens, this reaction is of a general nature and not especially characteristic in type. Infections by the purely pathogenic group, on the other hand, are characterized by certain distinctive features; such as, (1) the selective localization of the microbes, as, for example, meningococci in the meninges, pneumococci in the lungs, and typhoid bacilli in the lymph-adenoid tissue of the abdomen; and (2) the character of the lesion produced, as, caseation in tuberculosis, hepatization of the lung in pneumonia, and the ulcers of Peyer's patches in typhoid fever.

The invading microörganisms produce their effects either directly by mechanical means, or indirectly by the chemical substances which they produce. The presence of bacteria or other microörganisms in the tissues in great numbers causes more or less mechanical irritation just as any other foreign body does. Their luxuriant growth in the small blood-vessels may completely obstruct them. More frequently, however, the vessels in a focus of infection become plugged by the process of thrombosis. The tissues which are normally supplied with nourishment by the occluded vessels will suffer from lack of nutrition and actual necrosis may result.

The indirect effects due to chemical substances produced by microörganisms are of far greater importance in the process of infection than is the direct action of the microbes themselves. A few species of bacteria produce extracellular soluble toxins which, being absorbed, affect parts of the body at a distance from the primary focus of infection, as, for example, the action of tetanus toxin upon the central nervous system. The poisonous substances of most bacteria, however, are bound up in the bacterial cells and are only liberated upon the dissolution of the bodies of the microbes. In addition to these toxins, bacteria produce many other substances, acids, pigments, amides, chemical substances of the aromatic series, albumoses, peptones, enzymes and even gases, some of which may be important but little understood factors in infection.

The results of infection are both local and general. The local effect is an inflammatory reaction designed to rid the body of the infectious agents and to repair the damage done by them. At the site of the infection there occurs an exudate composed of (*a*) serum which brings into the area the bactericidal substances of the blood, dilutes the irritating and toxic bacterial products, and, when there is a free outlet, serves to mechanically wash the microbes out of the tissues; (*b*) fibrin, which aids by mechanically walling off the focus of infection, especially in the peritoneal cavity; and (*c*) leukocytes, which engulf and destroy the infecting microörganisms, and by their death and disintegration liberate proteolytic enzymes which liquefy necrotic tissues. While these exudative changes are still in progress, the reparative process begins, the destroyed tissues being replaced chiefly by the proliferation of the adjacent connective tissue. The processes just sketched are not specific and apply chiefly to infections by the group of "opportunist" parasites. In such specific infections as typhoid fever and tuberculosis, the local reaction shows certain characteristic deviations from that outlined above.

Bacteria may either remain localized in the immediate vicinity of the point of entrance, or they may spread, in several ways, from the primary focus of infection to more distant parts of the body. They may grow along a continuous mucous or serous surface, as when a rhinitis is followed by laryngitis, tracheitis, bronchitis and pneumonia. The microbes may be swept along in the lymph stream in the tissue spaces. Leukocytes that have engulfed bacteria may wander into lymph channels and themselves die and liberate living bacteria. Microbes in the lymph vessels may set up a lymphangitis; or they may reach the regional lymph-nodes and either be destroyed there or set up a lymphadenitis; or they may pass on in the lymph stream and finally reach the circulating blood. Bacteria may also enter the blood stream in fragments of infected thrombi, or by direct invasion of the blood, probably through the walls of capillaries, by a mechanism at present poorly understood. When bacteria enter the circulating blood they may be (*a*) quickly destroyed; or (*b*) survive long enough to reach other distant organs, lodge there, and set up new foci of infection; or (*c*) multiply in the blood stream and become established there causing septicemia.

The general effects of infection are characterized by changes in the blood, in the parenchymatous organs, in the temperature of the body, and in metabolism. Most infections cause an increase in the number of white blood cells, especially of the neutrophilic polymorphonuclear leukocytes in acute infections. In a few infectious diseases, like influenza, measles and typhoid fever, there is a leukopenia. Infections with animal parasites usually show an increase in the eosinophiles of the blood. In many infections, especially those associated with septicemia, there is a diminution in the number of red blood cells. The intravascular destruction of erythrocytes liberates hemoglobin which is the source of the icterus frequently observed in very severe infections, particularly with streptococcus. The coagulability of the blood may also be increased, as in pneumonia, or decreased, as sometimes happens in streptococcus infections. The blood-forming organs usually show changes, the yellow marrow of the long bones becoming redder in color. The spleen is usually enlarged, especially in those infections in which the microörganisms enter the blood stream in numbers. The endothelium of the capillaries, particularly in the serous membranes, is injured either by the toxic substances in the blood, or as a result of phagocytosis of bacteria by the endothelial cells, and petechial hemorrhages occur.

Toxic substances absorbed from the focus of infection cause degenerative changes in the parenchymatous organs, especially in those through which the poison is excreted, as, for example, the kidneys. In the acute infections, cloudy swelling and fatty degeneration of the heart, liver and kidneys are common. In the more chronic infections, amyloid degeneration may occur. Most infections are accompanied by more or less fever. This, is, in some instances, so characteristic as to be diagnostic. Infections also usually cause a greater or less deviation from the normal metabolism.

The final results of infection may be (1) rapid destruction of the microörganisms and complete recovery of the patient; (2) apparent recovery, with the attainment of a condition of latency by the invading microbes which later become active, causing either a recurrent attack of the disease or a localized inflammatory reaction at a distance from the original site of the infection; (3) the development of the "carrier state" in which the host, although in a healthy condition, still harbors in his body and discharges therefrom the microörganisms of the disease from which he suffered; or (4) the infection may become chronic; or (5) it may cause the death of the host.

BACTERIAL TOXINS

Bacteria produce poisonous substances of various kinds. The soluble poisons, or exotoxins, which are generally implied when the term "toxins" is employed, are definite products of bacterial metabolism and are present in the solution in which the bacteria are growing. Other bacteria are incapable of producing poisons of this kind, but carry in their bodies toxic substances which have been described as endotoxins. In addition to these types of bacterial poisons, many bacteria not pathogenic for animals, produce, by the decomposition of proteids or of lecithin, compounds some of which are powerful poisons. These are called ptomains.

Ptomains.—The nitrogen cycle is a series of changes occurring in nature whereby the complex proteid substances of plants and animals are built up from simpler nitrogenous compounds, and are again, upon the death of the plant or animal, reduced to the same simple nitrogen-containing substances, chiefly by saprophytic bacteria. At certain stages of the process of the reduction of the proteid molecule, less complex, non-specific compounds are formed, some of which are quite toxic. These substances were called ptomains by Selmi because they were found in decomposing dead bodies. The most important of these ptomains are nitrogen-containing substances, usually possessing an amino group and with or without oxygen. They are basic in reaction and have been called "animal alkaloids." The most poisonous ones that have been examined are found to contain oxygen.

Some ptomains are non-toxic. Others are violent gastro-intestinal irritants, others affect the central nervous system, and still others appear to act upon the nerve endings. It is doubtful if they are formed in the living body, certainly not in living tissues. Unless taken preformed in food, ptomains therefore probably have little to do with the actual causation of the symptoms of disease except in possible cases of absorption from gangrenous limbs.

Soluble Bacterial Toxins, or Exotoxins.—Bacillus diphtheriæ, Bacillus tetani, Bacillus botulinus, and possibly Bacillus pyocyaneus, produce regularly, as products of their normal metabolism, substances which are very poisonous, and are found free in solution in the medium in which the bacteria are growing. The exact chemical composition of these soluble toxins is not known. In the purest form in which they have been isolated, they still give tests for protein. Hence it is generally believed that they are either definitely protein in nature, or that they are so closely associated with the proteid molecule that forcible separation causes their destruction.

True bacterial toxins, when introduced parenterally into the animal body, induce the production of specific antibodies capable of neutralizing their poisonous properties. Exotoxins are, therefore, antigens. The antitoxin produced in response to injections of diphtheria toxin will neutralize that substance only, and not that of Bacillus tetani or Bacillus botulinus, and *vice versa*. In the possession of antigenic properties, these bacterial toxins are distinctly unlike ordinary poisons, such as alkaloids and inorganic substances, which do not induce antibody formation; and resemble certain vegetable poisons, like abrin and ricin, and animal poisons like snake venom, to which antibodies can be produced.

Bacterial toxins have been compared to enzymes with which they have certain attributes in common. Both deteriorate on standing; both are sensitive to the action of light; both are destroyed by heating to 80°C., or even to a lower temperature for a longer time; very small amounts of both produce disproportionately large results; and their injection into animals induces the formation of antitoxins or anti-enzymes, as the case may be, which render the corresponding toxin or enzyme ineffective. However, toxins are subject to the laws of quantitative combination much more definitely than are enzymes. Toxins, when once combined with the substance upon which they act, remain in combination, and are no longer active; while enzymes appear to enter into a temporary combination with the substrate and are liberated again, quantitatively and intact, when their action is complete.

When bacterial toxins are injected into susceptible animals, there is a definite incubation period before any symptoms manifest themselves. The time which elapses between the injection of the toxin and the appearance of the symptoms varies inversely with the size of the dose; that is, the larger the dose, the shorter the incubation period. But this incubation time cannot be brought below a certain irreducible minimum. Thus, mice, which are very susceptible to tetanus toxin, succumb to an ordinary lethal dose in about 36 hours. But 3600 lethal doses have been found to reduce the time to only 12 hours; while eight to nine hours appears to be the minimum time below which the period cannot be reduced in mice. In this important respect, also, bacterial poisons differ from ordinary poisons and snake venom, the action of which is prompt.

A wholly satisfactory explanation of this incubation period has not been suggested, although Ehrlich, Richet and de Waele have each offered such an explanation. Ehrlich thought that the action of toxin occurred in two stages; first, a union, perhaps chemical, of the poison with the susceptible cells, and second, the gradual injury of the cells by the toxin. According to Richet, the toxin is in an inactive state until rendered active by the body of the host. De Waele explains the incubation period on the physico-chemical basis of relative solubilities or of relative chemical affinities. The incubation time is that required for accumulation of the poison in the vulnerable tissues in which it may be more soluble or for which it has a special affinity.

The bacilli of diphtheria and of tetanus, the most typical examples of toxin-producing bacteria, lack invasive power. The growth of diphtheria bacilli is limited to an exudate or false membrane on a mucous surface, usually in the upper air passages, and from which its toxin is absorbed. Tetanus bacilli grow and produce their toxin in the subcutaneous tissues at the site of inoculation through a wound in the skin, and do not spread to any distance from this point. Bacillus botulinus, on the other hand, is unable to grow at the temperature of the human body. Hence its poison must be taken in preformed with food. Bacillus pyocyaneus, unlike the other bacteria which produce exotoxins, does possess invasive power of no small moment. Waite* has collected the literature and recorded cases of infection by this microbe. There is a just question whether Bacillus pyocyaneus should be classed with the bacilli of tetanus, diphtheria and botulism, on the basis of toxin formation. This bacillus produces a powerful proteolytic enzyme which diffuses into the medium in which it is growing. It is impossible to make experiments in phagocytosis with broth cultures of this microörganism, because the enzyme digests the leukocytes quite thoroughly.† Hence it is quite possible that the alleged exotoxin of Bacillus pyocyaneus is not a true soluble toxin but an enzyme which, when injected into animals, acts upon the body protein liberating poisonous substances.

The route of absorption of the toxin differs with the microörganism involved. The poisons of the bacilli of diphtheria and tetanus are harmless when taken by way of the stomach. The toxin of Bacillus botulinus, however, is absorbed from the gastro-intestinal tract in potent form. The toxin of Bacillus diphtheriæ is produced in the "membrane" in which it grows and is absorbed through the damaged epithelium either into the lymph or blood stream or into the neighboring nerve-endings or nerves. Tetanus toxin is absorbed along the nerve tracts. The classical experiments of Meyer and Ransom‡ showed, among other things, that, when tetanus toxin is injected into the hind foot of an animal, (1) the poison appeared in the sciatic nerve before it could be found in higher levels of the nervous system; and (2) severing the nerves to the limb prevented the development of the symptoms of tetanus.

The toxins of tetanus and botulism, and to a less extent, of diphtheria, have a special affinity for nerve tissue, the action of the first two being largely limited to the structures of the central nervous system. The injury to the central nervous system by tetanus toxin is either chemical or functional in nature, for no constant or characteristic gross or microscopic changes are found in the brain or spinal cord in persons dead of tetanus. The poison of botulism affects especially the centers in the brain stem, causing the characteristic clinical picture of bulbar paralysis. Changes in the Nissl bodies of the ganglion cells of this region with minute hemorrhages have been described in cases of botulism. Diphtheria toxin also has an affinity for nerve tissue, but its action is much more slow and the injury more gradual than in the cases of tetanus and botulinus poison. Peripheral degenerative changes in nerves appear to be responsible for the post-diphtheritic paralysis of the muscles of deglutition and for the paralysis of the heart that sometimes results in sudden death two or three weeks after an attack of diphtheria. In the acute stages of the disease, however, diphtheria toxin affects the parenchymatous organs, producing cloudy swelling and even fatty degeneration of the heart, liver and kidneys; injures the walls of the capillaries causing petechial hemorrhages into the pericardium and other serous membranes; and impairs certain centers, especially of the vagus and phrenic nerves, and perhaps the vasomotor center causing rapid and fatal fall in blood pressure. In guinea pigs, diphtheria toxin has a special affinity for the adrenal glands, causing edema, hyperemia, hemorrhage and even necrosis of the cells of the medullary portion. This lesion is not ordinarily found in cases of diphtheria in humans.

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* Waite, H. H., J. Infect. Dis., v (1908), p. 542.

† Simonds and Baldauf, J. Infect. Dis., vi (1908), p. 38.

‡ Meyer and Ransom, Arch. f. exp. Path. u., Pharmak., xlix (1903), p. 369.

The basis of the selective action of bacterial toxins on certain tissues has been sought on both chemical and physico-chemical grounds. Meyer and Overton's theory that the anesthetic properties of ether and chloroform are due to their ready solubility in the lipoidal substances of the central nervous system in which they accumulate in greater quantities than in other organs, and the later discovery that the danger of delayed chloroform poisoning increases in proportion to the amount of fat stored in the liver, have led to an explanation of the selective action of tetanus and botulinus toxins on the basis of the relative solubilities of these poisons in the intracellular substances of the central nervous system, as compared with their solubility in other cells. Our present knowledge of the chemical composition of these toxins does not furnish a foundation for this theory. There are other facts also that make this explanation doubtful. In the first place, the union between the toxin and the tissues for which it has an affinity is a firm one and is dissociated only with difficulty. It is for this reason that tetanus antitoxin is almost without effect if administered after symptoms of the disease have appeared. If the toxin were only in solution in the cells it would combine much more readily with antitoxin than if its bonds of affinity were already satisfied by chemical union with the intracellular substance. Hence it appears more probable that a positive chemical affinity, which results in a definite and firm chemical union between toxin and cell substance and a rapid quantitative disappearance of free and active toxin from the medium in which it reached the susceptible cells, is the cause of the selective accumulation of the bacterial toxins in certain cells, rather than mere physical differences in solubilities.

Ehrlich conceived each molecule of toxin to be composed of two parts, a haptophore group by which it becomes united with the cell; and a toxophore group which accomplishes the poisoning of the cell. The ready combination of the haptophore group with the intracellular substance for which it has a special affinity causes the disappearance of independent molecules of toxin as rapidly as they reach the susceptible tissues and this leads to their accumulation in these tissues. The causation of clinical symptoms is due to the action of the toxophore group, and this depends upon other factors, as for example, an optimum temperature.

The haptophore group of the molecule of tetanus toxin combines readily, even at low temperatures, with the substance of the central nervous system of the frog. But a higher temperature is necessary for the toxophore group to produce its effect. This is shown by the experiments of Meyer and Ransom. They found (1) that if frogs, injected with tetanus toxin, were kept at low temperatures the development of symptoms of the disease would be delayed for several weeks or would not occur at all, although the poison quickly disappeared from the blood and organs and was found only in the central nervous system during this period; (2) that frogs, so injected, and kept at incubator temperature, would develop tetanus within 24 hours or less; and (3) that similarly injected frogs, kept for a week at low temperature, would develop tetanus and die within about an hour after being placed in the incubator.

Soluble bacterial toxins are true metabolic products of the bacteria which produce them. The formation of toxin can be profoundly influenced quantitatively by the character of the medium in which the bacteria are growing. The poisons of Bacillus diphtheriæ and Bacillus tetani are formed in their greatest quantity when these organisms are growing in, and metabolizing, protein. However, the toxins are not of the nature of the poisonous "split products" of the protein molecule. Whether they are mere byproducts of bacterial metabolism or are substances of use to the microbes in their utilization of that part of the proteid molecule which they use in building up their own cell substance, cannot be determined at present. Theobald Smith was the first to show that in the presence of a fermentable carbohydrate the production of soluble toxin by the bacilli of diphtheria and tetanus, is reduced to a minimum. Bacillus botulinus was first found in sausage, and it seemed probable that its toxin might also be a product of the metabolism of protein. But the finding of botulinus poison in canned ripe olives, beets and asparagus makes it appear possible that the toxin of this microörganism may be a product of the metabolism of some substance other than protein.

Endotoxins.—When the washed bodies of bacteria are dissolved, substances that are highly toxic to animals are liberated. These substances are called endotoxins. They are rather universally present in the bodies of bacteria, both pathogenic and saprophytic. These endotoxins differ from the soluble exotoxins in several important respects. They are not found free in solution in the medium in which the microbes are growing; they are not specific for the bacteria from which they are derived; they are much more resistant to heat, that is, they are thermostable bodies; and they are devoid of antigenic properties. Their production does not appear to be greatly influenced by the change in bacterial metabolism induced by the addition of fermentable carbohydrate to the medium in which the bacteria are growing.

The exact nature of these endotoxins has not been positively determined. Whether they exist as definite substances preformed in the bodies of the bacteria and are merely liberated intact when the bacterial cells disintegrate; or whether they represent the poisonous part of the molecule of bacterial protein, is still in dispute. Their non-specific character is suggestive that they do not exist as distinct substances in the bodies of bacteria. The fact that Vaughan has obtained them by submitting bacterial protein to the same treatment that liberates a poisonous substance from egg albumin and horse serum, indicates that the so-called endotoxins are only the "poisonous split products" of bacterial protein. Furthermore, endotoxins resemble the poisonous portion of the ordinary proteid molecule (*a*) in their non-specific character; (*b*) in their lack of antigenic properties; and (*c*) in the similarity of their pharmacologic action. In proper doses, both produce effects similar to anaphylactic and peptone shock.

IMMUNITY

Much has been said in the preceding pages about resistance to infection. This is a relative term indicating the ability of the animal host to overcome invading microörganisms. The degree of resistance varies with the species, race and individual, and even in the same individual at different times. When the individual's resistance is below what may be considered normal, he is said to be *susceptible* to infection. An exceptionally high degree of resistance to infection is spoken of as *immunity* to that infection. Immunity is always relative and not absolute; its efficacy depends upon the virulence and dose of the infecting microörganisms. A degree of immunity that is capable of overcoming the moderate number of microörganisms that gain entrance to the body in ordinary spontaneous infections may not be able to cope successfully with the enormous numbers of virulent bacteria sometimes injected in the artificial or experimental production of disease, or that are occasionally taken into the body accidentally. Immunity to one species of microbe does not necessarily indicate a similar degree of resistance against another microörganism.

Immunity is of two kinds, natural and acquired. *Natural immunity* is a biologic character possessed by animals. It is congenital and is not based upon any previous event in the life of the individual. *Acquired immunity* is a marked increase in resistance to infection dependent upon the animal's having previously suffered from such an infection and recovered, or upon the employment of one or more of the means by which immunity may be artificially induced. The immunity which results from an attack of disease, for example, typhoid fever, is *spontaneously acquired immunity* that which follows the use of any of the known means of heightening resistance to infection is *artificially acquired immunity*.

Many of the known facts of natural immunity have been discussed under resistance to infection, and only need be mentioned here. One species of animal may be immune to a microörganism that is quite virulent and pathogenic for another species. Different species of animals may suffer from the same type of disease due to microörganisms remarkably specific for each species, as, for example, human, bovine and avian tuberculosis. This natural species immunity may be dependent upon differences in body temperature, in chemical variations in body fluids, or in the means of defense employed by the invaded host.

It is not so easy to find a plausible explanation for the high degrees of natural immunity frequently observed in different individuals of the same species. For in every epidemic there are found the extremely susceptible and the highly immune. No parallelism between the degree of natural immunity and the bactericidal power of the blood has been established. Furthermore, natural immunity cannot be passively transferred from one animal to another by the transference of blood or body fluids. This indicates that natural immunity rests upon some fundamental cellular difference in individuals of the same species, a difference that is probably physiologic rather than anatomical.

Artificial Immunity.—A single attack of many infectious diseases leaves the individual in a state of high resistance to a second infection with the same microörganism. This immunity may last throughout the remainder of the life of the individual. This is spontaneously acquired immunity. Certain other infections, on the other hand, may leave the victim in a condition of only temporarily increased resistance, or even in a state of heightened susceptibility to a second infection. The infectious diseases in which one attack is *usually* followed by a lasting immunity are bubonic plague, typhoid fever, cholera, small-pox, chicken-pox, scarlet fever, measles, yellow fever, typhus fever, syphilis and mumps. In the following diseases no lasting immunity is conferred by one attack; infections with the pyogenic cocci, gonorrhea, pneumonia, influenza, glanders, dengue fever, diphtheria, recurrent fever, tetanus, erysipelas, beriberi, malaria and tuberculosis.*

In all of the diseases of the first group the immunity remains long after the disappearance of any demonstrable increase of antibodies in the blood. In the case of the diseases of the second group, the ordinary laboratory tests do not show any abnormally low quantity of antibodies in the circulating blood. Hence the spontaneously acquired immunity must depend upon some change in the manner and speed of the reaction of the defensive powers of the cells of the body to a threatened invasion by the infecting microörganism.

The first successful attempt to intentionally produce a state of immunity by artificial means was that of Jenner in the case of small-pox. It had been observed that milkmaids who had acquired cow-pox by milking infected cows were immune to small-pox. By artificially infecting well persons with the virus of this disease of cattle he was able to protect them against the human disease. The method thus inaugurated came to be widely used under the name vaccination. Nearly a hundred years later Pasteur, using a method similar in principle, induced immunity to anthrax in sheep by injecting them with attenuated cultures of anthrax bacilli.

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* Zinsser, H., "Infection and resistance," 1st ed., N. Y. (1916), p. 60.

It is the work of Pasteur rather than that of Jenner that has led to the extensive use in modern times of the principle of vaccination for the production of artificial immunity. With the exception of small-pox, this application has been practically limited to bacterial infections. Pathogenic bacteria may be divided into the two groups of those having little or no invasive power but producing soluble exotoxins, and those which are able to invade the body but do not produce a soluble toxin. In the first group the immunity is against the products of the microörganisms; in the second it is against the microbes themselves. Hence, in the production of artificial immunity to diphtheria, tetanus and botulism, the animal to be immunized is inoculated with the bacteria-free, filtered, poisonous products of their growth in artificial media. To induce immunity to typhoid bacilli, streptococci and other members of the second group, a suspension of the bodies of the microörganisms themselves, washed free from their metabolic products, is injected. The injection of bacterial toxins stimulates the animal body to produce substances which circulate in the blood, and which have an affinity for, combine with, and neutralize the corresponding poisonous substance when they are brought together, either in the body of the animal or in a test tube. The injection of the bodies of the bacteria causes the animal organism to produce substances which destroy the homologous bacteria in one or another of the ways to be discussed later.

Artificial Production of Immunity to Bacterial Toxins.—In 1890 Behring produced in guinea pigs an antitoxic immunity against diphtheria. When it was learned, very soon thereafter, that the antitoxin in the serum of such an immunized animal would not only neutralize diphtheria toxin in a test tube, but possessed curative powers when injected into animals infected with diphtheria, the use of such sera in the treatment of human disease was begun. This method of inducing artificial antitoxic immunity is now used on a large scale in the commercial production of diphtheria and tetanus antitoxins Healthy horses are almost universally employed in the manufacture of these antitoxins. Clear, bacteria-free filtrates of broth cultures, in small, non-fatal, but gradually increasing doses are repeatedly injected subcutaneously at intervals of about seven days. A local reaction about the site of inoculation, and a general reaction characterized by a rise of temperature, follow these injections. Care is taken that both the local and general reactions have completely subsided before the next dose is given. After several weeks of this treatment, the serum of the horse is tested for its antitoxic potency.

When this reaches a sufficiently high degree, the animal is bled to the extent of 1 to 2 qt., the serum is allowed to separate and its antitoxic power is determined. This is the diphtheria antitoxin of commerce. The injections of the toxin are continued and the same horse can be used thus for many months or even years.

Active immunization against bacterial toxins has not been used to any great extent upon humans. Within the last few years, however, a method of active immunization against diphtheria has come into vogue by the use of a mixture of toxin and antitoxin, usually abbreviated to T.A. A small dose of diphtheria toxin, mixed with enough antitoxin to neutralize it, is injected subcutaneously in persons who have been exposed to the disease. Such an inoculation is followed by more or less reaction about the site of the injection. Not infrequently the local reaction is very severe and necrosis and extensive sloughing may result.

Antibacterial Immunity.—The active immunity produced by injections of the soluble toxic products of bacterial metabolism is directed against these substances only and not against the bacteria themselves. The antibodies so produced do not possess batericidal powers. But antitoxic immunity is not possible in the case of those microörganisms which do not produce soluble toxin. For immunizing against this group of bacteria, suspensions of the bacteria themselves are injected. For this purpose the suspensions may be composed of (*a*) sublethal doses of living, virulent bacteria; (*b*) attenuated living bacteria; (*c*) sensitized living bacteria; or (*d*) killed bacteria.

(a) Immunization with Sublethal Doses of Living, Virulent Bacteria.—In the discussion of infection it was stated that a certain minimum number of microörganisms must enter the body in order to produce infection. The injection of suspensions containing an accurately determined number of living tubercle bacilli has been used successfully by Webb, Williams and Barbour* in the immunization of guinea pigs against tuberculosis. Högyes used a similar method in immunization against hydrophobia, when he employed very small quantities of high dilutions of fully virulent virus. Such a method is always dangerous because of the variations in virulence of microörganisms, and differences in susceptibility of different individuals. Theoretically, this method of immunization is the most effective, but the associated dangers render it of little practical use.

(b) Immunization with Attenuated Living Bacteria.—This is, to all practical purposes, the method used by Jenner in immunization against small-pox, and by Pasteur in immunization against hydrophobia. A virus, whose virulence for humans is lowered by repeated passage through animals is, in the case of hydrophobia, still further attenuated by drying over sticks of sodium hydroxid. The longer the period of drying the greater the attenuation. Pasteur induced immunity to anthrax by the injection of attenuated cultures of anthrax bacilli grown at 40°C. This method of producing immunity is also not without danger. In human disease its use is practically limited to small-pox and hydrophobia.

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* Webb, Williams and Barbour, J. Med. Res., xx (1909), p. 1; xxiv (1911), p. 1.

(c) **Immunization with Sensitized Living Bacteria.**—The use of living microörganisms is the most effective method of producing anti-bacterial immunity. In order to obviate the dangers associated with the employment of sublethal doses of fully virulent and attenuated microörganisms, Besredka has recommended the use of suspensions of "sensitized" living bacteria. The living microörganisms are mixed with serum of an animal that has been immunized against that microbe. The mixture is allowed to stand for a time and then injected subcutaneously. The bacteria thus injected have been rendered "sensitive," that is more susceptible, to the destructive action of the offensive and defensive mechanisms of the animal into which they are injected. They are quickly destroyed and no infection follows, but immunity is developed. This method has been employed especially in active immunization against typhoid fever.

(d) **Immunization with Killed Bacteria.**—The use of suspensions of bacteria which have been killed either by heat or by means of chemical germicides, is the method most widely employed. Either method of killing the bacteria is attended by changes in their antigenic, or antibody-producing properties. The immunity induced by the injection of killed cultures differs quantitatively, and possibly qualitatively, from that which follows the use of living microbes. Care is taken not to heat the bacteria above the thermal death point of the particular microörganism against which immunity is desired.

A suspension of killed bacteria is commonly called a "vaccin or a "bacterin." If it is made from one of the ordinary stock cultures always kept in bacteriologic laboratories, it is called a "stock vaccin." If it is made from a culture of the microörganism isolated from the patient upon whom it is to be used, it is called an "autogenous vaccin." The latter is considered the more effective immunizing agent, especially in infections with certain bacteria like the streptococcus. This is due to the subtle differences in different strains of the same species of bacteria detectable only by serologic tests, and to the exquisite specificity of the antibodies which their injection calls forth.

ANTIGENS AND ANTIBODIES

In the discussion of immunity frequent use has been made of the terms "antigen" and "antibody." An antigen is any substance which, injected into an animal, induces in that animal the production of bodies which, when brought into contact with the corresponding substance, act upon it and cause chemical or physical changes in it. These induced bodies are called antibodies, and are definite reaction products of the animal organism.

Antigens are definitely proteid in nature. The only known exceptions to this statement are the toxic substances in mushrooms and poison ivy, which do not give reactions for protein, but to which Ford* has been able to produce antibodies. The fact that bacterial soluble toxins act as antigens may, perhaps, be taken as evidence of their proteid nature. Antigens are of two kinds, either clear homogeneous solutions such as serum, egg-white, or diphtheria toxin; or formed elements, like red blood cells and the bodies of bacteria.

The various antibodies, antitoxins, lysins, precipitins, agglutinins, opsonins, will be discussed in another place.

THEORIES OF IMMUNITY

The beginning of the scientific and experimental study of immunity dates from the investigations of Metchnikoff on the phagocytosis of minute solid particles and bacteria by certain cells in the water flea (Daphnia), reported in 1883. Applying these studies to the mechanism of resistance to infection, he made the rather broad generalization that the overcoming of the invading microörganisms was due to their engulfment and digestion by the phagocytes of the invaded host. A few years later, however, Nuttall showed that the cell-free serum of immunized animals possessed strongly bactericidal powers. A controversy arose between the adherents of the cellular or phagocytic theory, led by Metchnikoff, and those of the humoral theory, led by Pflügge and Buchner. During this controversy, Pfeiffer made an observation which at once became an important starting point in the further study of immunity. Pfeiffer† found that cholera vibrios, injected into the peritoneal cavity of a guinea pig immunized to these microbes, would, in a few minutes, swell, become granular, and finally dissolve and disappear. The same destruction occurred if the microörganisms were injected into a normal guinea pig along with a small amount of serum from an animal which had been immunized against cholera vibrios. This did not occur, however, if the vibrios were injected into a non-immune guinea pig either with or without non-immune serum. The leukocytes were found to play no part in the solution of the vibrios. This process came to be known as "Pfeiffer's phenomenon."

The modern conception of immunity harmonizes the old cellular and humoral theories. The immunity induced by the injection of different bacteria or their products does not always have the same mechanism. The antibodies resulting from immunization with the soluble bacterial toxins are chemical substances in solution in the blood serum or plasma; they combine with and neutralize the corresponding toxin without the interposition of phagocytes. The antibodies which result from the injection of an animal with the bodies of bacteria, like typhoid bacilli, act upon the bacterial cell and cause its solution or lysis, without the aid of leukocytes. Both of these instances are examples of the humoral theory because phagocytic cells have no immediate part in the process. The destruction of the pyogenic cocci, on the other hand, is due chiefly to their engulfment and digestion by polymorphonuclear leukocytes. The immunity to these microörganisms, induced artifically or acquired spontaneously, is dependent, not upon any stimulation of the production or of the activity of these leukocytes, but upon the presence in the serum in increased quantity, of an antibody called opsonin which unites with the cocci and prepares them for phagocytosis. Hence, in this form of immunity the actual destruction of the microbes depends upon two factors, a humoral element, opsonin, and a cellular element, phagocyte.

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* Ford, W. W., J. Infect. Dis., iii (1906), p. 191; iv (1907), p. 541.

† Pfeiffer, Ztschr. f. Hyg., xvii (1894), p. 355; xviii, p. 1.

The modern conception or theory of immunity may be summed up in a single sentence: In artificial immunity to all bacteria and bacterial toxins, a soluble antibody or humoral element, is the essential factor; in one type of infection, this soluble antibody may alone destroy the microbes or neutralize their poison; in another type, the final destruction of the microörganism depends upon the *additional* action of a phagocytic or cellular element.

Origin and Nature of Antibodies.*—Ehrlich's Side-chain Theory.—Antibodies are of two kinds: (1) Those which enter into combination with their corresponding antigen without producing any visible change in the mixture, as, for example, the combination of diphtheria toxin and antitoxin which leaves the solution clear and unchanged; and (2) those whose union with their antigens results in a physical change that is visible even to the naked eye, as, for example, erythrocytes and bacteria dissolve and disappear leaving a clear solution when acted upon by a lytic serum; or a precipitate is formed in a previously clear solution by a precipitating serum; or bacteria or other cells may congregate in clumps and settle out of a suspension when acted upon by an agglutinating serum.

The origin of antibodies in the animal organism has been sought for by many workers but no satisfying positive facts have been discovered. There is considerable evidence to indicate that the blood forming organs—bone marrow, lymph-nodes and spleen—are the probable site of the greatest quantitative production. In the early stages of immunization, antibodies have been found in greater amount in the spleen than in the blood. However, removal of the spleen has not been followed by any evidence of lowered resistance to infection. Repeated injections of benzol, which cause serious injury to the bone marrow and produce a marked leukopenia, greatly diminish the production of antibodies.† When the antigen has been injected into the peritoneal or pleural cavities, there has seemed to be a greater quantity of antibodies in the fluid in the cavity so injected than elsewhere in the body. This apparent local immunity may mean that the formation of antibodies is a function of all of the tissues or organs of the body; or, as Zinsser suggests, of endothelial cells in general.

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* Hektoen, L., Origin and Fate of Antibodies, Harvey Society Lecture (Jan. 15, 1910).

† Simonds and Jones, J. Med. Res., xxxviii (1915), p. 197; Hektoen, J. Infect. Dis., xix (1916), p. 69.

The nature of antibodies has been studied most extensively by Ehrlich and his pupils. He has formulated an elaborate theory which explains most of the important facts of the humoral aspects of immunity. Ehrlich's theory of immunity grew out of his conception of the mechanism of the nourishment of protoplasm. He conceived the molecule of protoplasm to be composed of a central or functional atom group, surrounded by other atom groups or side-chains, which have an affinity for nutritive substances. These affinity-groups he called receptors. A molecule of toxin or other antigen combines with the cells of the body by the cell receptors, that is, by the same sort of mechanism by which nutriment is taken into the cell. A receptor to which a molecule of toxin has attached itself is no longer useful to the cell and is thrown off. In replacing receptors thus lost, an excessive number is produced and the excess receptors are cast off without their affinities satisfied. These free receptors are the antibodies found in the blood serum. There are thus three steps, according to Ehrlich's theory, in the process of the formation of antibodies; the fixation of the toxin, the over-production of receptors, and the liberation of the excess receptors in the circulating blood.

Just as antigens differ from each other in their physical and chemical characteristics, so the receptors, and the antibodies which they form, also differ from each other. Ehrlich recognized three orders of receptors and antibodies. Those of the first order have a single unsatisfied combining group which unites molecules of relatively simple nature without producing any profound physical change, as for example when antitoxin combines with and neutralizes toxin. Receptors and antibodies of the second order have a combining or affinity group which unites with the antigen, and a zymotoxic group which works a physical or other change in the antigen, as, for example, when a serum acts upon bacterial cells and causes them to come together in clumps, or when it prepares bacteria for phagocytosis. Those of the third order, called amboceptors, have two combining groups, one of which unites with the antigen, the other combines with an enzyme usually called complement or alexin. This complement is a separate body which exists preformed in the serum and is not a specific product of the process of immunization. Lysins which cause the solution of cells, are antibodies of the third order. That group which unites the amboceptor to the antigen is called the haptophore, or grasping, group, and is the specific part of the antibody. It will unite only with antigen of the same kind as that used to induce its formation. The complementophile group is non-specific, and the same complement can combine with many amboceptors.

Ehrlich's theory not only explains more of the known facts of immunity than any other theory that has been advanced but it has served as an effective stimulus to research in the field of immunity. However, numerous objections have been advanced against it. In the first place, it does not explain the immunity that evidently resides in the fixed cells of the body, because an immunized animal may withstand a larger dose of toxin than could be neutralized by the antitoxin in the blood. Furthermore, this theory presupposes that the power of producing antibodies is possessed by all the cells of the body because all cells must possess receptors for nutrition. This being true, one would expect that the place of greatest antibody formation would be in those tissues for which a given toxin has the greatest selective affinity. There is nothing at present to indicate that the central nervous system is the place of origin of tetanus antitoxin. Again, it is difficult to understand how an animal, whose serum already contains large quantities of antitoxin, can be stimulated to produce more of this same antibody by the injection of the corresponding toxin. For it would seem that the toxin so injected would combine with the free cell receptors in the blood and thus be prevented from reaching the cells to stimulate the production and extrusion of more receptors to form antitoxin. In spite of these objections, Ehrlich's theory continues to be the most satisfactory one yet advanced.

Passive Immunity

In the immunity thus far discussed, the animal being immunized produced its own antibodies in response either to a spontaneous infection, or to the injection of microbes or their soluble toxins. The greatly increased resistance to infection was dependent partly upon the antibodies circulating in the blood, and partly upon changes in the character and rapidity of the reaction on the part of the body cells themselves. It was found by Behring that the serum of one animal immunized against diphtheria toxin would protect another animal against an infection with the bacilli of diphtheria and an injection of many times the lethal dose of diphtheria toxin. This is *passive immunity.* An animal thus immunized is not injected with antigen, toxins or bodies of bacteria, but with the serum of another animal which has been actively immunized by the injection of antigen. It does not itself produce the antibodies which neutralize the toxin, but receives them preformed in the serum of another animal that has produced them.

The passive transference of immunity has been most successfully employed as a therapeutic measure in infections with bacteria that produce soluble exotoxins. It has not been so successful in those infections in which the destruction of the bacteria themselves is desired. In other words, toxin-neutralizing substances can be passively transferred in immune serum; bactericidal antibodies, however, cannot be so effectively conveyed to another animal, or when conveyed, are not able to protect the animal so thoroughly as is an antitoxic serum.

Passive immunity is applied in a practical way in the treatment and prevention of diphtheria and tetanus. The use of diphtheria antitoxin has reduced the death-rate from this disease by from 50 to 70 per cent. of the pre-antitoxin mortality. Park found that it requires from 12 to 18 hours for the absorption into the blood of the greater part of the antitoxin from the subcutaneous tissues. Hence in malignant cases of diphtheria, intravenous injections are necessary.

In the prevention of diphtheria in persons exposed to the disease, from 200 to 1000 units of antitoxin are injected. The passive immunity thus transferred lasts only about two weeks, and the immunizing dose must be repeated if the danger is still imminent. Tetanus antitoxin is used as a preventive with more success than as a curative agent after the development of the disease. The union between the tetanus toxin and the vulnerable tissues has become so firm by the time symptoms appear, that the antitoxin cannot break up the combination and free the cells of the toxin.

ANTITOXINS

Antitoxins are antibodies which are produced in response to the injection of certain poisonous substances, and have the power of neutralizing the corresponding poison. The antitoxin consists of free receptors in the serum. They have been produced for the soluble toxins of the bacilli of diphtheria, tetanus and botulism; for the vegetable poisons, abrin and ricin; for snake venom; and for the poisons of certain mushrooms and of poison ivy.* Diphtheria antitoxin is the only official therapeutic serum in the United States. The method of standardizing it is established by law and its oversight is placed in the hands of the United States Public Health Service.

The toxin is prepared by growing diphtheria bacilli in slightly alkaline broth, containing not more than 0.1 per cent. dextrose, in wide, shallow flasks to permit an abundance of oxygen. After about seven days, the culture is passed through a procelain filter, and the clear filtrate is the toxin used for immunization. The potency of the toxin is determined and it is also employed in the standardization of the antitoxin. A unit of toxin is that amount which will kill a guinea pig weighing 250 gm. in about four days, and is spoken of as an M.L.D., or minimum lethal dose. The method of immunizing horses in the manufacture of diphtheria antitoxin has already been discussed. When the process of immunization has reached a proper stage the horse is bled and the serum collected. It is necessary to determine the neutralizing power or potency of each lot of the serum. Its potency is expressed in units, a unit of therapeutic antitoxin being that quantity necessary to completely neutralize 200 M.L.D.'s of *pure* diphtheria toxin. The *official unit* in the United States is "the neutralizing power possessed by an arbitary quantity of diphtheria antitoxic serum kept under special conditions to prevent deterioration in an authorized laboratory." This standard measure is dried diphtheria antitoxin which is kept in the Hygienic Laboratory of the United States Public Health Service in Washington, D. C. It is preserved in sealed vacuum tubes under the influence of pentaphosphoric acid, and in a cold place protected from light. This standard antitoxin is supplied to the makers of diphtheria antitoxin in the form of a glycerinated solution of such strength that each cubic centimeter contains just one "official unit" and will neutralize one M.L.D. of *pure* toxin. Standard antitoxin is kept because it is less complex and more stable than is diphtheria toxin.

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* Ford, W. W., J. Infect. Dis., iii (1906), p. 191; iv (1907), p. 541.

For the standardization of a given lot of new antitoxin, it is first necessary to determine the potency of the toxin to be used by comparing it with the standard antitoxin. This is done by injecting subcutaneously into guinea pigs of 250 gm. weight, varying mixtures of toxin and standard antitoxin. The following table, abbreviated from Rosenau,* will illustrate the method:

1 immunizing unit 0.15 c.c. toxin = no reaction.
1 immunizing unit 0.16 c.c. toxin = very slight hyperemia at site of injection, *i.e.*, L_0 dose.
1 immunizing unit 0.17 c.c. toxin = apparent reaction.
1 immunizing unit 0.19 c.c. toxin = hyperemia and edema at site of injection, and late paralysis.
1 immunizing unit 0.20 c.c. toxin = sometimes death in five or six days, sometimes only late paralysis.
1 immunizing unit 0.21 c.c. toxin = always death about the fourth day, *i.e.*, L_+ dose.
1 immunizing unit 0.22 c.c. toxin = acute death, second or third day.

From this experiment it is learned that one unit of standard antitoxin just neutralizes 0.16 c.c. of the toxin. This is the L_0 dose, that is, the amount of toxin which is just neutralized by one antitoxin unit so that no local or general reaction results. This experiment also shows that 0.21 c.c. is the amount of toxin which will neutralize 1 unit of standard antitoxin and cause the death of a guinea pig weighing 250 gm. in four days. This is called the L_+ dose.

The potency of a given lot of antitoxin is next determined by injecting guinea pigs weighing 250 gm. each with mixtures of the L_+ dose with varying quantities of the serum to be tested, as illustrated in the following table:*

0.21 c.c. toxin 1/150 c.c. serum = no effect.
0.21 c.c. toxin 1/200 c.c. serum = late paralysis.
0.21 c.c. toxin 1/250 c.c. serum = death on fourth day.
0.21 c.c. toxin 1/300 c.c. serum = death on second day.

From this experiment it is seen that 1/250 c.c. of the serum contains antitoxin sufficient to neutralize the toxin in the L_+, or test dose, leaving just enough to kill a standard guinea pig in the standard time of about four days. Such a serum would contain 250 units of antitoxin in each cubic centimeter.

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* Rosenau, M. J., Hygienic Lab. Bulletin, No. 21 (1905).

A unit of diphtheria antitoxin was defined in terms of "pure" diphtheria toxin. Because of the readiness with which the toxin deteriorates, a "pure" form is difficult to obtain. There is no fixed or constant relation between the toxicity of crude diphtheria toxin and its power to neutralize antitoxin. In the illustration cited above, 0.21 c.c. of toxin was the L_+ dose, which represents 2 M.L.D's. of toxin one of which neutralized the unit of standard antitoxin, the other caused the death of the animal. Theoretically, then 0.105 c.c., or one-half of the L_+ dose, should contain 1 M.L.D. But it was found in the first cited experiment, that 0.16 c.c. of toxin was absorbed by the immunization unit of antitoxin, while only 0.05 c.c. additional was necessary to kill the guinea pig. The reason for this discrepancy is that the crude toxin contains bodies that are not toxic but that are still capable of uniting with and neutralizing antitoxin. Ehrlich designated these bodies as toxoids, and believed them to be molecules of toxin that had lost their toxophore or poisoning group, while retaining their haptophore, or grasping or attaching group. It is because of the presence of changing amounts of these toxoids in crude toxin that it is necessary to preserve a standard antitoxin, rather than a standard toxin.

The rate of reaction between toxin and antitoxin is always a relatively slow one and depends upon the concentration, the temperature, the medium, the presence of inorganic salts, etc. When the combination has once taken place, it is a firm union and is not easily broken up. For this reason antitoxin must be given early in diphtheria infection, because the union between toxin and receptors still attached to the cells is as firm as that between the toxin and the free receptors that constitute the antitoxin. Largely because of the firmness of this union, Ehrlich* believed it to be of the nature of a chemical reaction between two strong reagents, like hydrochloric acid and sodium hydroxid. On the other hand, because of the slowness and apparent incompleteness of the reaction, Arrhenius and Madsen† have attempted, without undisputed success, to apply to the toxin-antitoxin reaction the well known chemical law of mass action. They conceive that this reaction is a reversible one, and is more like that taking place between a weak acid and a weak base.

The antitoxin in a serum appears to be closely associated with globulin fraction. Methods of concentrating diphtheria antitoxin depend upon the precipitation of the globulin of the serum, which brings down with it the antitoxin. The chief benefit arising from concentration of an antitoxic serum is the possibility which it furnishes of injecting smaller quantities of foreign protein. The use of concentrated antitoxin reduces the danger of untoward results following its injection in exactly the same proportion as the total volume of foreign protein is reduced.

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* Ehrlich, P., "Collected Studies on Immunity," New York (1906), p. 547.

† Arrhenius, "Immunochemistry," New York (1907).

AGGLUTININS

Agglutinins are antibodies which cause the corresponding bacteria or other cells to come together in clumps. They have an affinity for the agglutinable substance of bacteria, that is, for the agglutiongens, which are the substances in the bacterial cells that stimulate the production of agglutinins in spontaneous infections or in artificial immunization. Different species, and different strains of the same species, of bacteria differ in the quantity of agglutinable substance which they possess, and, therefore, in the quantity of agglutinin which they induce. Typhoid, paratyphoid and colon bacilli and cholera vibrios are rich in agglutinogen and injection of these microörganisms usually results in the production of high agglutinating power in the serum. Paratyphoid bacillus "A" yields a stronger agglutinating serum than does paratyphoid bacillus "B." Bacillus mucosus capsulatus and Bacillus welchii appear to be very poor in agglutinable substance, and animals immunized against them do not readily yield a highly agglutinating serum.

Cells other than bacterial also contain agglutinogens. Agglutinating sera may be produced for red blood cells. Normal serum contains small quantities of agglutinins for various bacteria and for the erythrocytes of other animals.

Agglutinins, like antitoxins, are closely associated with the globulin fraction of the serum, and they are precipitated with it.

Agglutinins are antibodies of the second order, that is, they possess a haptophore group which unites with the agglutinable substance of the cells, and a zymophore group which induces the clumping. The mechanism of this clumping has been studied and various theories have been advanced to explain it. The theory elaborated by Bordet* has largely replaced the others. Bordet believes that the agglutinin fixes itself to the agglutinable substance of the bacteria by a process of adsorption, and this causes a change in the surface tension of the organism. The bacteria then congregate in clumps, just as particles in a colloidal suspension come together in the "breaking" of an emulsion. This probably is the result of the action of the cations of the salt, present in the solution, upon the cells that have had their charge of negative electricity reduced by the union with the agglutinin. Agglutination will not take place in salt-free solutions.

An agglutinating serum heated to 75 or 80°C. loses its power to cause bacteria to clump, but is still capable of combining with the bacteria and of using up the available receptors of the agglutinable substance of the cells. Agglutinins that have been thus changed are called agglutinoids. Bacteria that have bound agglutinoids cannot be made to clump either by adding fresh normal serum containing complement or by adding unheated, active agglutinating serum. The receptors of the cells have been occupied by the agglutinoids, whose zymophore groups have been destroyed by heating.

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* Bordet, Ann. de l'Inst. Pasteur, xiii (1899), p. 225.

Agglutinins are only partially specific antibodies. The serum of an animal immunized against typhoid bacilli will contain agglutinins for this microörganism and also for colon and paratyphoid, that is, for related, bacilli. These are called group agglutinins. The agglutinins for the bacteria used in immunization are the primary or homologous, those for the related species are the secondary or heterologous, agglutinins. Group agglutination is believed to be due the possession by bacterial cells of several agglutinogens, some of which are held in common with other related species. The primary or homologous agglutinins can be determined in two ways. In the first place it is present in greater amount than the secondary antibody, and the serum will agglutinate the homologous microörganism in higher dilutions than the related bacteria. In the second place, an absorption experiment (Castellani's reaction) may be made. This depends upon the fact that when agglutinin unites with bacterial cells it is used up and disappears from the solution. The homologous bacteria will absorb all of the agglutinin from a solution of such a serum, while the related bacteria will take out only that part which will unite with its common agglutinable substance.

Agglutinins are not bactericidal substances. Bacteria which are clumped will, after a time, separate and continue to multiply. However, it works some change in the microörganisms, for typhoid bacilli tend to grow out in long threads in typhoid agglutinating serum.*

Although not bactericidal, agglutinins may play an important part in the overcoming of infection. Bull† found that the injection of a pneumococcus-agglutinating serum into rabbits suffering from a pneumococcus septicemia caused an almost immediate disappearance of the cocci from the circulating blood. They were found in clumps in the capillaries of the liver, spleen and lungs where they were taken up in great numbers by phagocytes. Those pneumococci which escaped agglutination and remained free also escaped phagocytosis for a much longer time. Pneumococci injected into the ear vein of a rabbit without any immune serum also quickly disappeared from the blood stream in the same manner, due apparently to the small quantity of agglutinin present in the normal serum of the animal.

Agglutination has been employed for the practical purposes of aiding in the diagnosis of disease, of identifying unknown bacteria, and of classifying different strains of the same species of bacteria into groups. For the diagnosis of disease, dilutions of the patient's serum are added to suspensions of a known bacterial culture. If clumping of the microörganisms occurs in relatively high dilutions of the serum, this is an indication that the patient either is suffering, or has previously suffered, from an infection with the microbe which his serum agglutinates, or has received injections of a "vaccin" made from the corresponding microörganism. For the identification and classification of bacteria, a suspension of the unknown microörganisms is tested against the serum of an animal which has been immunized against a known species of bacteria. Agglutination of the unknown organism by the known serum serves to identify it. Agglutination is used especially for the diagnosis of typhoid and paratyphoid infections; and for separating the types of paratyphoid and dysentery bacilli and pneumococci and streptococci.

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* Pfaundler, Centralbl. f. Bakt., I Abt., xxiii (1898), pp. 9, 71, 131.

† Bull, C. G., Jour. Exp. Med., xxiv (1916), p. 7.

Isohemagglutinins and Blood Grouping.—It has long been known that the serum of some animals contains natural lysins and agglutinins for the erythrocytes of animals of a different species. In 1902 Landsteiner* observed the isoagglutination of human red blood cells; that is, the agglutination of human erythrocytes by human serum. He separated three groups according to the interrelationship between the agglutinability of the corpuscles and the agglutinating power of the serum. Later Hektoen† showed the presence of a fourth group. The discovery of isohemagglutination explained the disastrous results that had been previously been observed occasionally to follow the transfusion of a patient with the blood of a healthy individual.

In 1907 Jansky‡ arranged the four groups mentioned above in the order of frequency of their incidence. His paper was published in an inaccessible, not widely read journal and did not attract the attention that it deserved. Later Moss§ arranged the four groups in a more arbitrary manner; and his classification has been followed in most of the literature on blood grouping published in America. A committee of the American Association of Pathologists and Bacteriologists, however, has recommended the universal adoption of Jansky's classification on the grounds of priority and because of its more logical arrangement of the groups. Jansky's classification may be summarized as follows:

Group I.—Serum agglutinates the corpuscles of Groups II, III and IV.
Cells not agglutinated by the serum of any group.
Group II.—Serum agglutinates the cells of Groups III and IV.
Cells agglutinated by serum of Groups I and III.
Group III.—Serum agglutinates the cells of Groups II and IV.
Cells are agglutinated by serum of Groups I and II.
Group IV.—Serum agglutinates cells of no group.
Cells agglutinated by serum of Groups I, II, and III.

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* Landsteiner, Zt. f. Med., iii (1902).

† Hektoen, J. Infect. Dis., iv (1907), p. 297.

‡ Jansky, Klin. Sbornik, viii (1907), p. 85.

§ Moss, Bull. Johns Hopkins Hosp., xxi (1910), p. 63.

The differences between the classifications of Jansky and Moss are shown in the following table.

TABLE I.—SHOWING THE DIFFERENCES BETWEEN THE JANSKY AND MOSS CLASSIFICATION OF BLOOD GROUPINGS

Cells	Jansky grouping				Moss grouping			
	Serum				Serum			
	I	II	III	IV	I	II	III	IV
I....................	−	−	−	−	−	+	+	+
II....................	+	−	+	−	−	−	+	+
III....................	+	+	−	−	−	+	−	+
IV....................	+	+	+	−	−	−	−	−

+ = Agglutination. − = No agglutination.

From this table it is seen that Groups I and IV are reversed in the two classifications. It is therefore of very great importance that the classification used in grouping any individual be stated. Group IV of Moss has been called the "universal donor group" because the corpuscles of members of this group are not agglutinated by the serum of members of any other group. Disaster would follow the use of an individual of Jansky's Group IV as a "universal donor." Groups II and III are the same in both classifications. This is shown also in the following table in which the incidence of the several groups is given.

TABLE 2.—SHOWING THE INCIDENCE OF THE SEVERAL BLOOD GROUPS IN THE JANSKY AND MOSS CLASSIFICATIONS

Group	Jansky grouping,* per cent.	Moss grouping,† per cent.
I..............................	42.84	3.1
II..............................	41.38	42.4
III..............................	10.36	8.3
IV..............................	5.42	46.2

* Karsner and Ecker, "The principles of immunology," Philadelphia (1921), p. 100.

† Karsner, J. Am. Med. Assoc., lxx (1918), p. 769.

The grouping as seen in adults is rarely present at birth, and does not usually become fully established until about the end of the first year of life.* The corpuscles acquire agglutinophile receptors before the serum contains agglutinins. That is, the properties which determine the grouping become established in the corpuscles earlier than in the serum. Isohemagglutinins are said to be present in the mother's milk. Once established, the grouping of any given individual remains permanent. Karsner thinks that some chronic diseases may cause quantitative but not qualitative changes in the isohemagglutinins of the serum.

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* Happ, J. Exp. Med., xxxi (1920), p. 313.

V. Dungern and Hirschfeld, and Gichner have presented evidence that human isoagglutinogens are inherited according to the Mendelian formula for a dihybrid. The unit characters are the presence of isohemagglutinins as dominant and their absence as recessive. Because of the hereditary transmission of group characteristics attempts have been made to use blood grouping for the medicolegal purpose of establishing parentage. The contradictory results obtained by Buchanan* on the one hand, and by Ottenberg† and by Gichner‡ on the other, render this method as yet of questionable value for this purpose.

Among the numerous theories that have been advanced to explain the mechanism of isohemagglutination, that of Landsteiner, which has been confirmed by Koeckert,§ is the most satisfactory. The existence of the four groups is due to a difference in distribution in the serum and cells of two agglutinins (designated *a* and *b*) and two agglutinogens (designated *A* and *B*), respectively. These are distributed as follows in Jansky's classification.

Group	Agglutinin (Serum)	Agglutinogen (Corpuscles)
I	*a b*	— —
II	— *b*	*A* —
III	*a* —	— *B*
IV	— —	*A B*

The serum of Group I (Group IV, Moss) possesses both agglutinins and, therefore, agglutinate the corpuscles of all of the Groups except its own; the cells of this Group lack both agglutinogens and are, therefore, not agglutinated by any serum. The serum of Group IV (Group I, Moss) lacks both agglutinins and therefore does not cause the clumping of the cells of any of the Groups; while the cells of this Group, possessing both agglutinogens, are clumped by the serum of any group except the homologous one. Groups II and III each possess only one agglutinin and one (heterologous) agglutinogen.

It is evident from the distribution of agglutinins and agglutinogens shown above, that the four Groups not only do not exhaust the mathematical possibilities of the distribution of these bodies, but do not even exhaust the biologic possibilities. For example: It is possible to have patients whose corpuscles may contain either agglutinogen but whose serum contains no agglutinin; and *vice versa*. Guthrie and Huck* have recently described such unusual combinations. The incidence of these unusual distributions of agglutinogens and agglutinins is probably less than that of Jansky's rare Group IV. But the fact that they do exist, and the readiness with which they may be overlooked by the methods of group determinations commonly used, render them a very real danger in blood transfusions.

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* Buchanan, J. Amer. Med. Assn., lxxviii (1922), p. 89; and lxxix (1922), p. 180.

† Ottenberg, J. Immunol., vi (1921), p. 363; J. Amer. Med. Assn., lxxix (1922), p. 2137.

‡ Gichner, J. Amer. Med. Assn., lxxix (1922), p. 2143.

§ Koeckert, J. Immunol., v, (1920), p. 529.

Blood grouping has its practical application in the selection of suitable donors for tranfusions, and of skin for grafting. It is obviously safer to use a donor belonging to the same Group as the recipient. When the patient belongs to one of the less common Groups, for example, Group IV (Group I of Moss), the difficulty of finding a donor of the same Group may render it necessary to employ a "universal donor" belonging to Group I (Group IV, Moss). The corpuscles of this Group are not agglutinated by the serum of any Group. But the serum of this Group will cause clumping of the cells of all of the other Groups. However, the great dilution of the serum of the transfused blood in the blood of the recipient reduces the danger of intravascular agglutination to a minimum. Nevertheless, the possibility of quantitative differences in the amount of agglutinin in the serum of different members of Group I (Group IV, Moss) renders the use of "universal donors" not without a modicum of danger.

As an added precaution in pernicious anemia the patient's serum should be tested directly against the corpuscles of the prospective donor even though he belong to the same Group as the patient. The serum of some patients with pernicious anemia contains a strong lysin for corpuscles belonging to the same blood Group.

Mention has already been made of the fact that the grouping of an individual is not fully established until about the end of the first year of life, and in the serum later than in the corpuscles. For this reason, a donor of any Group may be used with reasonable safety for transfusing an infant under six months of age.

Schawan† found that "isografts of skin obtained from donors of the same blood Group as the recipients or from Group IV (Moss) donors became permanent takes and grew almost if not equally as well as autografts. Isografts where the recipients were of different groups did not remain as permanent growths except when Group IV (Moss) skin was used or when the recipient was a member of Group I (Moss). Group I (Moss) recipients grew permanent skin grafts from donors from all four groups and apparently equally well. Group IV (Moss) skin grew permanently on recipients of all groups, but only Group IV (Moss) grafts and autografts remained as permanent takes on Group IV (Moss) recipients. Therefore it may be assumed that skin grafting obeys the principle of blood grouping."

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* Guthrie and Huck, Bull. Johns Hopkins Hosp., xxxiv (1923), pp. 37 and 80.

† Schawan, Am. J. Med. Sc., clvii (1919), p. 503.

PRECIPITINS

Kraus found that the serum of rabbits immunized against typhoid bacilli and cholera vibrios would give a precipitate when mixed with clear filtrates of old broth cultures of these microörganisms. Further experiments have shown that all proteins contain precipitinogens that induce the formation of antibodies which cause a precipitate to form when added to a clear solution of the corresponding protein. Thus, if the serum of species *A* is injected into an animal of species *B*, the serum of *B* will come to contain specific precipitins for the serum of species *A*. However, an animal will not produce precipitins for the serum of another individual of its own race. Precipitins, like agglutinins, are antibodies of the second order, and are associated with the globulin fraction of the serum. It has been suggested that precipitation by specific precipitins and agglutination are one and the same process under different conditions; one occurring in perfectly clear homogenous solutions, the other in suspensions of cells; one being due to the action of an antibody upon very minute particles in colloidal solution, the other to its action upon larger solid particles in suspension. But the precipitate formed is insoluble and cannot be dissolved by mineral salts nor by weak sodium hydroxid. Furthermore, the precipitate is composed chiefly of constituents of the antiserum and not of the antigen. In this respect precipitins differ from all of the other antibodies, in that their effect is produced upon the antibody and not upon the antigen.

Precipitins are specific in the same sense that agglutinins are, that is, they produce precipitates in the presence of higher dilutions of their corresponding antigens than in the presence of related antigens. Groups precipitins are formed however. For example: Anti-human serum will produce precipitates in high dilutions of human serum and in low dilutions of the serum of monkeys and the anthropoid apes. The precipitin test has been used by Nuttall* in an extensive study of blood relationships.

The precipitin test† has found practical application in the identification of blood stains for medicolegal purposes. Blood that has been dried on garments or surfaces for years will still give a precipitin reaction with its specific anti-serum. In pure food work, this test has been used to detect horse meat in sausage.

OPSONINS

In 1895 Denys and Leclef showed the importance of the presence of serum in the causation of phagocytosis of bacteria. They believed, however, that the serum acted by stimulating the leukocytes, rather than upon the bacteria themselves. About 10 years later, Wright and Douglas called special attention to the subject, discovered the correct explanation of the phenomenon, and gave the name *opsonin* to the antibody that is concerned in the reaction. They found that opsonins act upon the microörganisms and not upon the leukocytes. They "substitute a positive for a negative chemotaxis," and cause some change in the bacteria that makes easy their engulfment by polymorphonuclear leukocytes.

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* Nuttall, "Blood relation and immunity," Cambridge University Press (1904).

† L. Hektoen, Jour. Amer. Med. Assoc., lxvi (1906), p. 1407. Full literature.

Non-specific opsonins are present in normal serum, that is, such serum will prepare limited quantities of a large variety of bacteria for phagocytosis. These normal opsonins are therefore of importance in the destruction of bacteria that invade the body and doubtless play a significant rôle in natural resistance to infection. The opsonic power of the serum of an animal can be increased by the injection of killed cultures of many bacteria, especially of the pyogenic cocci. The opsonins induced by this process of immunization are called immune opsonins and differ in several important respects from those found in normal serum. Normal opsonins are thermolabile bodies and are destroyed by heating to 55 to 60°C.; immune opsonins are thermostable bodies and resist much higher temperatures. The former will fix and remove complement from serum; the latter do not and are antibodies of the second order. Normal opsonins, also, are neutralized by weak solutions of certain chemical substances, such as calcium chlorid, potassium sulphate, formalin and lactic acid, which do not affect immune opsonins. Normal opsonins are, therefore, rendered inactive by the same means that destroy complement.*

The mechanism of the action opsonins is not known. They become fixed to the bodies of bacteria but do not cause the death of the microbes. This is evident from the fact that an opsonin-containing serum may not be bactericidal; and that leukocytes which have engulfed more bacteria than they can destroy, not infrequently die themselves and liberate living microörganisms. This is one of the means by which infection spreads to distant parts of the body. Hektoen found that anthrax bacilli grew readily in dog serum, but were quickly destroyed by a mixture of dog serum and leukocytes.

Lysins

The antibodies thus far discussed do not alone cause the death of bacteria. Antitoxins neutralize and render harmless bacterial poisons but do not affect the microörganisms themselves. Agglutinins cause bacteria to congregate in clumps from which they may later grow again. Opsonins unite with bacterial cells which are destroyed only if they are taken up by phagocytes. Bacteriolysins, however, are antibodies which, without the aid of leukocytes, cause destruction and solution of bacteria for which they have an affinity. They are formed during the progress of certain spontaneous infections, especially in typhoid fever and cholera; and may be induced artificially by repeated injections of bacterial cells. Many bacteria, like typhoid bacilli and cholera vibrios, are rich in lysinogenic substances; others, like staphylococci and streptococci, contain relatively little lysinogen.

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*L. Hektoen, Jour. Amer. Med. Assoc., lxvi (1906), p. 1407.

Pfeiffer found that the serum of an animal immunized against cholera vibrios protected a nonimmune animal against infection if the immune serum and the vibrios were injected into the peritoneal cavity together. In the peritoneal cavity the microörganisms were rapidly dissolved and destroyed. But the immune serum did not cause the death of the vibrios when mixed with them in a test tube. Hence, the body of the animal supplied something that was necessary to render the serum effective. It was later discovered that vibriolysis could be induced in vitro if the immune serum was used when quite fresh or if fresh normal serum was added to it. It was about this time that Bordet* discovered that repeated injections of one animal with the erythrocytes of another resulted in the production of hemolysin which would cause the laking of blood cells from the same species as those originally injected. The action of hemolysins is so much more readily detected and measured than that of bacteriolysis, that the greater part of our knowledge of lysins in general is due to the study of hemolysins.

Lysins are antibodies of the third order. They have two combining groups, one of which unites with the antigen, the other with a ferment-like body, or complement also present in fresh serum. The process of lysis, whether of bacterial or other cells, is the result of the interaction of two substances the immune body or amboceptor, and the complement or alexin. These substances or bodies posssess very unlike characteristics. Neither can, alone, bring about the solution of cells. Antigen, amboceptor and complement are sometimes spoken of as a lytic system. If erythrocytes are the antigen, we have a hemolytic system.

The amboceptor is the specific part of the system, that is, it will only unite with cells like those used to induce its formation. It possesses two groups, a cytophile group with an affinity for the receptors of its homologous cells, and a complementophile group with an affinity for complement. Normal serum contains a small quantity of amboceptor. The function of the amboceptor is to bring an increased amount of complement into union with the cell. Whether such a process causes the death of a bacterial cell depends ultimately upon its sensitiveness to the action of the particular complement present. Dog serum normally contains anthrax amboceptor but no complement to which this microörganism is susceptible. Fresh rabbit serum does contain the necessary complement, so that the combined action of the normal amboceptor of dog serum and the complement of rabbit serum quickly dissolves and destroys anthrax bacilli.

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* Bordet, Studies in immunity, collected by Gay, New York (1907).

Amboceptor is relatively thermostable and is not destroyed by heating to 55 to 60°C. Amboceptor will unite with its corresponding cell at 0°C. It may thus be removed from fresh serum without disturbing the complement which does not combine at this temperature. This combination between cell and amboceptor is, within limits, a reversible one. Red blood cells can take up more amboceptor than is necessary for their lysis. If these overcharged cells are washed free of serum, mixed with untreated cells of the same kind, and the mixture allowed to stand for an hour, the added cells will take up amboceptor from the others; upon the addition of complement to this mixture all of the red cells will suffer lysis.

Complement is non-specific and is found in all fresh serum. It is not increased in the process of active immunization. It is thermolabile and is rendered inert by heating to 55 to 60°C. It is also inactivated by exposure to light, by shaking, and by the action of dilute solutions of many salts, like calcium chlorid, and sodium oxalate and citrate. Like toxins, the complement contains two groups, a haptophore group, by which it attaches itself to the amboceptor, and a toxophore group which produces its destructive effect. Hence, it is necessary to differentiate the combining and the toxic actions of complement. Complement may be without effect either because it does not combine with the amboceptor, or because its toxic power is lost. Heat appears to destroy the toxophore group without affecting the haptophore group. It then becomes complementoid and is still able to attach itself to amboceptor, but cannot produce its characteristic effect.

The origin of complement is not known. Metchnikoff's view that it is derived from leukocytes has been disproved. It does not appear to come from the tissues of the body, for these have the power of fixing complement *in vitro*. Its nature is equally obscure. It is known only as a property of fresh serum. Kyes* found that lecithin would serve as complement for the hemolysin of snake venom. Although the presence of lipoids has been found to influence the action of complement, it cannot be identified with lipoids.

The unity of complement has been disputed. Bordet believes that there is only one complement that is present in all fresh serum. Ehrlich thought that there is a multiplicity of complements which differ among themselves in the affinities of their haptophore groups and in the action of their toxophore groups. This is the more widely accepted view among serologists.

The combination of antigen, amboceptor and complement takes place quantitatively so that the amount of each component necessary to cause complete lysis of a given quantity of red blood cells can be accurately measured. This titration can be made easily in a hemolytic system and is done daily in certain routine laboratory tests. This does not mean, however, that cells cannot, in the presence of an excess of amboceptor, combine with more than is necessary to cause lysis of the cells.

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* Kyes, P., Berl. kl. Wchnschr., Nos. 38 and 39 (1902).

Since the discovery of Pfeiffer's phenomenon it has been known that bacteriolytic sera are capable of protecting an animal against infection with the corresponding microbe if the serum and microörganisms are injected together. This protective action is, however, very limited. Anti-bacterial sera, unlike antitoxic sera, do not obey the law of multiples. For instance, 100 times the amount of lytic serum that protects against one lethal dose of homologous bacteria, will not protect against 100 lethal doses. For this reason the curative results from the therapeutic use of anti-bacterial sera have not equalled those derived from antitoxic sera. The reasons for the relative inefficiency of antimicrobic serum are chiefly of a speculative character. The microörganisms may be in tissue spaces that are inacessible to the lytic bodies in the serum in sufficient quantities to cause destruction of the microbes. The available complement of the body may be insufficient in quantity or unsuitable in kind. Finally, "deviation" or "deflection" of the complement may occur. In the presence of a limited quantity of complement and a great excess of amboceptor, the homologous cells may escape destruction because most or all of the available complement becomes attached to free-floating amboceptors and is "deviated" of "deflected" from those attached to the cells.

Complement Fixation. The Wassermann Reaction

The combination of antigen plus antibody possesses a strong affinity for complement which becomes fixed in the combination and is removed from the solution. This "fixation of complement" must not be confused with the entirely different phenomenon of "deviation or deflection of complement" defined in the preceding paragraph. The principle of fixation of complement was first described by Bordet and Gengou. They mixed suspensions of bacteria (B. pestis, B. typhosus, etc.) with complement and the serum of a horse which had been immunized against the corresponding microörganisms. After incubation this mixture showed no visible change. But that an important change had taken place was made evident by adding to the mixture a suspension of red blood cells and a homologous hemolytic serum. No hemolysis occurred showing that the complement had been firmly fixed by some interaction between the antigen (the bacilli) and the antibody in the immune serum. This fixation did not occur when a normal serum or a heterologous immune serum was substituted for the homologous immune serum. The red blood cells and the corresponding hemolytic serum served as an indicator by which the binding of the complement was made visibly evident.

This reaction has been found to be specific within about the same limits that the precipitin reaction is specific. Neisser and Sachs found that there are group complement fixing bodies just as there are group precipitins. But they were able to show, for example, that anti-human serum was capable of fixing complement in much higher dilutions in the presence of human serum as antigen, than with monkey serum as antigen.

The nature of the complement fixing body and ts relation to precipitin, agglutinin and amboceptor (lysin) have been extensively investigated. Complement fixation will occur in a higher dilution than will precipitation. This does not, however, necessarily prove that the complement-fixing body and the precipitin are separate and distinct but merely that a visible precipitate is not essential to complement fixation. It has been suggested that the immune molecule or antibody has more than one combining complex. One of these may combine with the antigen and produce a precipitate, and another may enter into a combination with antigen and complement and bind or fix the latter. It is also possible that the concentration of the various ingredients may affect the results. Dean found that the optimal relationship between antigen and immune serum for the production of a precipitate may be different from the optimal concentrations for complement fixation. Zinsser thinks that precipitation is merely a colloidal phenomenon which may or may not coincide with the phase of the greatest complement fixation according to other fortuitous conditions which may favor or retard flocculation of colloids. From the information now available it does not appear possible to make a dogmatic statement in regard to the identity of the precipitin and the complement-fixing body.

Much the same obscurity attaches to the relation of complement fixation to lysis. Karsner* thinks that Zinsser's view of precipitation may also be applied to amboceptor (lysin). Lysis of cells may be an incident in complement fixation, certain conditions favoring lysis, others merely complement fixation.

Numerous conditions and substances interfere with complement fixation and may be serious sources of error in the practical application of this phenomenon in the diagnosis of disease. The optimal concentration of sodium chlorid solution for fixation of complement is that which is isotonic with blood serum. Fixation does not occur in salt-free solutions. Too great concentrations of salt also inhibit the reaction, probably by a deleterious effect on the complement itself. Numerous other substances also interfere with the reaction, for example, sodium fluoride, sodium sulphite, boric and benzoic acids, formalin, many lipoids (cholesterin, lecithin, etc) in sufficient concentration, organic colloids like gelatin or glycogen, and inorganic colloids like kaolin and carbon. "Acids and alkalis in considerable concentration permanently destroy complement, but if the injury is due to dilute alkali the complementary activity may be restored by neutralization." Tissue extracts and body fluids, especially pathologic exudates (ascitic and arthritic fluids) are also anticomplementary. Blood serum kept for more than a few days develops anticomplementary power. The latter fact has a practical importance in the use of this phenomenon in the diagnosis of disease.

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* Karsner and Ecker, "The principles of Immunology," Philadelphia (1921).

When the fixation of complement was found to be a specific reaction, attempts were made to give it a practical use in the diagnosis of disease. Bordet employed it for diagnosing typhoid fever. Neisser and Sachs used this method to indentify blood stains for medicolegal purposes without finding it to have any advantages over the precipitin test long used for this purpose. Fixation of complement has also been made use of in the diagnosis of gonorrheal arthritis and tuberculosis. Results in these diseases have been somewhat discordant, especially in tuberculosis. In this disease difficulty has been encountered in producing a suitable antigen. The most extensive use of the principle of fixation of complement has been in the diagnosis of syphilis, commonly known as the Wassermann reaction.

Since its first announcement in 1906, by Wassermann, Neisser and Bruck the test has undergone numerous modifications, especially in regard to the antigen used and the hemolytic system employed as indicator. Wassermann first used a watery extract of the liver of a syphilitic fetus, which Schaudinn had recently shown to be teeming with the treponema of syphilis. It was later learned that alcoholic extracts, not only of syphilitic fetal liver, but of normal human liver and heart, and of guinea pig and beef heart made a perfectly satisfactory antigen. Perhaps the most commonly used antigen now employed for the Wassermann test is an alcoholic solution of the acetone-insoluble portion of beef heart or guinea pig heart. The addition of cholesterol to this acetone-insoluble alcoholic extract seems to increase its antigenic value.

Various hemolytic systems have been used as indicators. Sheep corpuscles and antisheep amboceptor are probably most commonly employed. Human and chicken red blood cells and the homologous amboceptor have also been successfully used.

For the actual performance of the Wassermann test it is essential that the antigen, amboceptor and complement be accurately titrated. The amboceptor and complement are titrated against 1 c.c. of a standard (usually a 2.5 per cent.) suspension of corpuscles homologous with the amboceptor to be used. The smallest amount of amboceptor and of complement that will cause complete hemolysis of 1 c.c. of such a standard suspension is 1 "unit" of amboceptor and complement respectively. In making the test, two units of each of these ingredients are used.

The titration of the antigen is somewhat more complicated. For this purpose two rows of small test tubes are set up. In one set of tubes increasing dilutions of the antigen are mixed with two units of complement and 0.2 c.c. of a known positive serum; in the second set of tubes, the same dilutions of antigen are mixed with two units of complement and 0.2 c.c. of a known negative serum. These are incubated for one-half hour at 37.5°C. and then two units of amboceptortor and 1 c.c. of standard suspension of corpuscles are added and the mixture again incubated for one-half hour. The first row of tubes will reveal the smallest quantity of the antigen that is capable of binding complement in the presence of known positive serum; the second row will show the minimum amount of antigen that is itself anticomplementary. There should be a considerable difference between these two minima. The smallest amount of antigen capable of binding complement in the presence of known positive serum is called one unit. In the performance of the test, two units of antigen are used.

In order to avoid the many sources of error that are inherent in the Wassermann test, it is very necessary to make numerous controls. These controls should include, as a minimum, (*a*) control of the anticomplementary action of the antigen in the strength used; (*b*) control of the fixing power of the antigen in the strength used in the presence of known positive serum; (*c*) control to determine whether the patient's serum is itself anticomplementary; (*d*) a control to insure the avoidance of the phenomenon of complement deviation in case an excess of amboceptor was used.

The Wassermann test is not, strictly speaking, an immunologic reaction, because substances, such as alcoholic extracts of guinea pig and beef heart, that have no relation whatever to the treponema pallidum, may be successfully used as antigen. The most generally accepted explanation of the mechanism of this reaction is that there occurs a precipitation of the globulin of the serum by the lipoidal colloids of the antigen and adsorption of the complement by the precipitate.

The Wassermann test is not a "specific test," for syphilis in the sense that the Widal reaction is "specific" for typhoid fever. This fact does not, however, detract materially from its value in diagnosis. The percentage of positive reactions in cases with a clinical diagnosis of syphilis varies with the worker and with the stage of the disease. Karsner* has tabulated the results of Boaz and of Craig in over 12,000 cases which illustrate both of these points.

Clinical diagnosis	Boaz's cases			Craig's cases		
	Number cases	Positive	Per cent. positive	Number cases	Positive	Per cent. positive
Primary syphilis	1060	629	59.0	908	813	89.5
Secondary syphilis	3526	3181	90.0	1889	1917	96.1
Tertiary syphilis	1212	1029	84.1	638	558	87.4
Early latent syphilis	983	504	51.0			
Late latent syphilis	1520	695	39.0			
Tabes dorsalis	159	115	72.0	173	790	67.3
Paresis	405	402	99.2	28	25	89.2
Congenital syphilis	....	....	....	22	7	68.1
	8865	6555	73.8	4658	4010	86.2

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* Karsner and Ecker, "The principles of immunology," Philadelphia (1921).

The use of the Wassermann test has tended to modify opinion as to the validity of Colles' Law. Many mothers of syphilitic infants, who have no symptoms of syphilis themselves, do give a positive Wassermann. On the other hand, Williams* found 43 such mothers whose Wassermann reaction was at all times negative. From his very wide experience Williams thinks that the question of the validity of Colles' Law should be regarded as *sub judice.*

ANAPHYLAXIS

In 1904 Theobald Smith† observed that guinea pigs that had been used once in standardizing diphtheria antitoxin very frequently died suddenly after receiving, at a later time, a second injection of an amount of horse serum that was harmless to a normal animal. Flexner had observed a somewhat similar phenomenon in dogs that were given repeated injections of a foreign serum. This phenomenon of hypersensitiveness to the parenteral injection of foreign protein has been termed "anaphylaxis," a name invented by Richet. Later v. Pirquet used the term "allergy" to describe the "altered reaction" that followed the injection of a foreign protein into a sensitized animal, the reaction sometimes observed after an injection of a large dose of diphtheria antitoxin, the different type of reaction that follows a second vaccination against small-pox, etc. It is evident that the conditions included under the name "allergy" have certain elements in common but are by no means identical either in their mechanism or manifestations.

Attempts have been made to introduce a satisfactory classification of these phenomena. The common element in all of these reactions is an increased susceptibility or hypersensitiveness, that is, a specific and peculiar reaction, with characteristic symptoms, "to the administration of or contact with a quantity of a substance which, to the majority of the members of the same species of animal, is innocuous."

Doerr‡ groups all of these reactions under the general term "Allergy" and subdivides them as follows:

I. To non-antigenic substances.
  1. Hypersensitiveness (drug idiosyncrasies).
  2. Hyposensitiveness.

II. To antigenic substances.
  1. To toxins.
    (*a*) Hypersensitiveness, not due to antibodies.
    (*b*) Hyposensitiveness, due to antibodies.
  2. To proteins.
    (*a*) Hypersensitiveness due to antibodies—as anaphylaxis.
    (*b*) Disappearance of hypersensitiveness—anti-anaphylaxis (not due to antibodies).

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* Williams, Bull. Johns Hopkins Hosp., xxxi (1920), p. 335.

† Smith, J. Med. Research., xiii (1905), p. 341.

‡ Doerr, "Handbuch d. path Organismen," Kolle and Wassermann, 2nd ed., cxii, p. 947.

This classification has two disadvantages. It introduces the phenomena of hyposensitiveness that have a different etiology from the corresponding opposite condition; and by making the possession or lack of antigenic properties by the exciting substances the basis of subdivision etiologically related phenomena are separated and placed in different subgroups.*

Coca and Cooke† have formulated a more elaborate and satisfactory classification. They include all of the related specific and peculiar reactions under the general term Hypersensitiveness, which is defined as "a susceptibility in man and animal that is mediated by a special mechanism which may be specifically influenced (toward increased or diminished sensitiveness) by suitable administration of the exciting agent." Their classification is as follows:

Hypersensitiveness.
  I. Normal (Allergy).
    1. Dermatitis venenata.
    2. Serum disease,—ordinary.
  II. Abnormal.
    1. Anaphylaxis. (*a*) Exhibited only against precipitinogenic proteins; (*b*) is passively transmitted to normal animals with the serum of sensitized animals; (*c*) is completely removable by the procedure of desensitization; (*d*) is not inherited; (*e*) is expressed, in the acute reaction, in symptoms and pathologic changes which differ *characteristically* in the guinea pig, rabbit and dog.
    2. Hypersensitiveness of infection. (*a*) Is exhibited against non-precipitinogenic substances, e.g., tuberculin; (*b*) is not passively transferable; (*c*) is not inherited; (*d*) is expressed in the acute general, as well as in the cutaneous, reaction by symptoms and pathologic changes which do not differ characteristically in different animal species; (*e*) and regularly develops in the course of an infection.
    3. Atopic hypersensitiveness. (*a*) Is exhibited against both precipitinogenic and non-precipitinogenic substances; (*b*) is not passively transferable; (*c*) is inherited, subject to the dominant gene; (*d*) is expressed in pathologic changes and symptoms, the more important of which differ from anaphylactic shock.

Symptoms resembling anaphylactic shock may be produced by the intravenous injection of non-sensitized animals with various substances, such as peptone, agar, etc. These reactions are frequently spoken of as anaphylactoid.

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* Coca, "Tice's practice of medicine," Philadelphia, i (1923), p. 108.

† Coca and Cooke, J. Immunol., viii (1923), p. 163.

The chief facts of anaphylaxis were first clearly presented by Rosenau and Anderson,* and may be briefly stated as follows: A single injection of a small and harmless dose of a foreign proteid into an animal renders it hypersusceptible to a second injection of a small dose of the same protein after an interval, or incubation time, of at least 10 days. The minimum amount of protein that will sensitize is exquisitely small, for example, one-millionth of a cubic centimeter of horse serum (Rosenau and Anderson) and one twenty-millionth of a gram of crystallized egg albumin (Wells). This state of hypersensitiveness is of long duration. Properly made injections of the homologous protein during this period are usually fatal.

The injection of any protein that is foreign to the body or blood of an animal, except those lacking in tryptophan and tyrosin, such as gelatin, will render the animal hypersensitive to that protein. The specificity depends upon the chemical composition of the protein and not upon its biologic relations.† Two proteins of rather widely different biologic sources may sensitize to each other if they are closely related chemically; while two other proteins, of similar biologic origin may not sensitize to each other.

For the production of the anaphylactic reaction in a sensitized animal, the second dose of protein must be administered parenterally, and may be given as subcutaneous, intravenous, intrathecal or intracerebral injections. The subcutaneous route gives the least uniformly successful results, probably because of the relatively slow absorption of the protein from the subcutaneous tissues. It is essential that at least a certain minimum quantity of the protein be brought into the circulation within a minimum of time. A relatively small amount injected rapidly into a vein will produce a profound reaction; while a relatively large quantity, injected very, very slowly at first, may be without effect.

The reaction thus produced is called anaphylactic shock. It is extraordinarily spectacular in the suddenness of its onset and in the violence of its symptoms. It differs strikingly in its clinical manifestations in different animals. For a given species of animal, however, the character of the reaction is constant, regardless of the protein used for inducing it. In the guinea pig, the initial symptom is usually a scratching of the nose and mouth. This is followed by a rapidly increasing dyspnea, and the animal dies of suffocation. The characteristic finding at autopsy is an enormous inflation of the lungs with air; they do not collapse when the chest is opened. This condition was shown by Auer and Lewis‡ to be due to a tetanic contraction of the secondary and tertiary bronchioles whose walls, in this animal, contain an exceptional quantity of smooth muscle tissue.

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* Rosenau and Anderson, Hygienic Lab. Bull., No. 29 (1906); No. 36 (1907).

† Wells and Osborne, J. Infect. Dis., viii (1911), p. 66; xii (1912), p. 341; xiv (1914), p. 364 and 377; xvii (1915), p. 259.

‡ Auer and Lewis, J. Exp. Med., xii (1910), pp. 151 and 638.

It is more difficult to produce anaphylactic shock in the rabbit. In this animal, the initial symptom may or may not be characterized by violent excitement. Ultimately, the animal lies on its side and, without evidence of dyspnea, gives a few gasps and dies with the eyes bulging in exophthalmos. There is first a slight rise, and then a marked fall in arterial blood pressure. At autopsy the lungs are collapsed; the right side of the heart is distended with blood, the left side almost empty. The underlying physiologic reaction in the rabbit was shown by Coca* to be an obstruction in the pulmonary circulation due to a tetanic contraction of the abundant muscular coat of the pulmonary arterioles, an anatomical arrangement that is apparently peculiar to the rabbit.

In anaphylactic shock in the dog there is a rapid, marked and prolonged fall in arterial blood pressure; a simultaneous, marked fall in venous pressure (superior vena cava) of similar duration; a simultaneous, pronounced and prolonged rise in the pressure in the portal vein; an increase in the volume of the liver; an increased flow of lymph from the liver and splanchnic area; leukopenia; and a loss of coagulability of the blood. At autopsy the chief finding is a marked venous stasis in the liver and branches of the portal vein. Weil estimated that 61 per cent. of the total blood of the body was impounded in the liver of a dog in anaphylactic shock. That the liver is of primary importance in anaphylactic shock in the dog has been shown by Manwaring† Deneke, and by Voegtlin and Bernheim.‡ If the liver is excluded from the circulation by an Eck fistula, the animal does not become sensitized (Deneke). Complete exclusion of the liver from the circulation, or an Eck fistula with temporary occlusion of the hepatic artery, entirely prevents the production of an anaphylactic shock in a sensitized dog.

The two chief objects of investigations of anaphylactic shock have been to determine (*a*) the source and mechanism of the formation of the alleged anaphylactic poison; and (*b*) the locus and mode of its pharmacologic action. The numerous theories that have been advanced to explain the mechanism of anaphylactic shock may be classified as humoral and cellular. The humoralists may be further separated into two groups: (1) Those who derive the poison from the injected foreign protein; and (2) those who derive it from the injected animal's own serum.

Vaughan§ conceived the protein molecule to be composed of a central poisonous nucleus or "archon" which is common to all proteins; and additional chemical groups which, by their number and arrangement about the "archon," confer upon a protein its specific nature. The first or sensitizing injection of protein, Vaughan believes, induces the production of enzymes which are capable of "splitting" the homologous protein molecule and liberating its poisonous portion. For Vaughan, then, the anaphylactic poison is the "archon" or poisonous nucleus common to all proteins, liberated as a result of the action of a proteolytic enzyme specific for the protein used in sensitization. The injection of protein "poisonous split products," produced artificially, induces in animals symptoms very similar to those that characterize anaphylactic shock in that animal.

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* Coca, Proc. Soc. Exp. Biol. and Med., xvi (1919), p. 47.

† Manwaring, Zt. f. Immunitation, viii (1910), p. 1.

‡ Voegtlin and Bernheim, J. Pharmacol. and Exp. Therap., ii (1911), p. 507.

§ Vaughan, V. C., "Proteid split products in relation to immunity and disease," Philadelphia (1913).

Friedberger* believes that the sensitizing dose of protein induces the formation of precipitins. The precipitate that is produced in a precipitin reaction is known to absorb complement. He obtained a poison capable of causing the manifestations of anaphylactic shock by incubating the inactivated serum of a sensitized animal with an excess of complement and homologous (sensitizing) protein. He called this poison "anaphylatoxin," and derived it from the action of complement upon the combination of specific precipitin and precipitinogen (sensitizing protein).

Jobling, and Novy believe that the anaphylactic poison is formed from the animal's own serum; but they differ in their theories as to the mechanism of its formation. Jobling's† view may be briefly stated thus: In the blood of every animal there exist protein-splitting enzymes and anti-enzymes in a state of approximate dynamic balance. The injection of a second dose of homologous protein into a sensitized animal results in a sudden reduction of the anti-enzyme. This permits the unchecked action of the proteolytic enzyme which liberates a relatively large quantity of the digestion products of the animal's own serum protein. These poisonous products, which Jobling calls "sero-toxins" are closely related to, if not identical with, the anaphylatoxin of Friedberger. Because this "serotoxin" can be produced *in vitro* by shaking up the animal's own serum with kaolin (process of adsorption) or with chloroform (fat solvent), Jobling believes that the unsaturated fatty acids of the serum constitute the anti-enzyme.

Novy‡ was able to produce typical symptoms of anaphylactic shock in non-sensitized animals, (1) by the injection of the animal's own serum which had been allowed to stand in contact with trypanosomes for a time; (2) by the intravenous injection of dilute solutions of agar-agar; and (3) by the intravenous injections of distilled water. He concluded that the anaphylactic poison is derived from the animal's own body proteins by the action of an "inducing body." This body is the result of an antigen-antibody combination. The specificity of the reaction depends upon the production of this "inducing body," which may also be brought about by such substances as bacteria, kaolin and agar-agar.

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* Friedberger, Zt. f. Immunitätsforsh, ix (1911), p. 394; xiii (1912), p. 127.

† Jobling and Peterson, J. Exp. Med., xx (1914), p. 468; xxii (1915), p. 401.

‡ Novy and DeKenif, J. Inspect. Dis., xx (1917), pp. 499–854; J. Am. Med. Assn., lxviii (1917), p. 1524.

There are two serious objections to all of these humoral theories of the production of a soluble toxin that circulates in the blood. In the first place, no such soluble poison has been demonstrated in the blood of an animal in the state of anaphylactic shock. Furthermore, all known ferments and immune bodies require an incubation period before the products of their action are liberated. The spectacular promptness with which the symptoms of shock follow the injection of a homologous protein into a sensitized animal would seem to effectually preclude the possibility that either ferments or antibodies are concerned in this reaction.

Vaughan,* Friedberger† and some others believe that anaphylactic shock results from the action upon the injected protein of enzymes or antibodies that are free in the circulating blood. It has appeared to some investigators, however, that the sensitizing dose of protein is relatively too small to be adequate to induce the formation of receptors in sufficient numbers to be cast off by the cells in which they are formed. Hence Pearce and Eisenbrey‡ Schultz,§ Manwaring and others believe that the anaphylactic poison is produced from the injected protein by the action of "sessile" receptors which are still attached to the cells. Pearce and Eisenbrey transfused a dog with the blood of two normal dogs in order to wash out as thoroughly as possible any circulating antibodies; and found that the sensitized animal still gave an anaphylactic reaction when injected with the homologous protein.

Weil‖ discarded the anaphylatoxin theory. He was convinced that, in the guinea pig at least, anaphylactic death is due to a cellular reaction, and that the serum plays no rôle therein. In anaphylactic shock in the dog, alterations do occur in the chemical composition of the blood. These changes are due to the liberation of protease as a result of the reaction of the sensitized cells to the antigen. Weil seemed to be convinced that the activity of the protease in the circulating blood was an accompaniment and not the cause of the anaphylactic shock. The anaphylactic reaction, then, was an expression of the alteration of cellular equilibrium which results when the external antigen is brought into contact with the cellular antibody.

Placing the locus of antigen-antibody reaction within the cell, puts it beyond the possibility of detection by any methods now available. However, the absence of any incubation period in the initiation of anaphylactic shock when a sensitized animal is injected with the homologous protein casts serious doubt upon the validity of any theory that explains the reaction upon the basis of the action of sessile receptors. Dale* has formulated a physical conception of the reaction. "All that is needed," says he, "is that the antibody should have such a specific physical relation to the antigen that when the two meet, a disturbance of the condition of colloidal solution is set up in the muscle fiber."

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* Vaughan, V. C., "Proteid split products in relation to immunity and disease," Philadelphia, (1913).

† Friedberger, Zt. J. Immunitätsforsch, ix (1911), p. 394; xiii (1912), p. 127.

‡ Pearce and Eisenbrey, J. Pharm. and Exp. Therap., vi (1912–1913), p. 21.

§ Schultz, Hygienic Lab. Bull., No. 80 (1912).

‖ Weil, J. Med. Res., xxvii (1913), p. 497; xxx (1914), pp. 87 and 299; J. Immunol., ii (1917), p. 469.

Schultz,† Weil and others have shown that unstriated muscle of a sensitized animal reacts very strongly to "anaphylactic poison." In view of the differences in the manifestations of anaphylactic shock in different animals and the anatomical basis therefor, as described above, we may formulate a tentative general principle for the explanation of the underlying physiology of this reaction. The clinical manifestations and postmortem findings in anaphylactic shock in a given animal are largely determined by the species or individual peculiarities in the distribution of smooth muscle in the body. Animals that have exceptionally large amounts of non-striated muscle in their bronchi or bronchioles will show the guinea-pig type of shock. Its presence in excessive amount in any particular vascular bed will result in an interference with the flow of blood through that organ. Whether the prolonged contraction of this muscle and the resulting obstruction to blood flow will result fatally, will depend chiefly upon the position of the organ in the circulatory system. Contraction of the relatively large amount of smooth muscle in the walls of the rabbit's pulmonary arteries causes a marked fall in systemic blood pressure and dilatation of the right side of the heart. The enormous impounding of blood in the liver of the dog in anaphylactic shock, together with the rise in portal pressure and the fall in arterial and venous (vena cava) pressures, suggested the possibility that there might be a similar peculiarity in the hepatic vein of this animal. Simonds and Arey‡ studied the hepatic veins in some 21 species of animals and found that the dog is peculiar in the possession of a truly enormous quantity of smooth muscle in the wall of the hepatic vein. This finding seemed to furnish an anatomical basis for the manifestations of anaphylactic shock in the dog, and to make possible the formulation of a uniform theory, such as outlined above, to explain the physiologic mechanism of anaphylactic shock in the guinea pig, rabbit and dog.

Manwaring,§ Peterson‖ and their co-workers have taken exception to this view. Both of these investigators seem to consider that the symptoms of anaphylactic shock in the dog are due to an increased permeability of the capillaries, especially those of the liver, with rapid loss of fluid from the blood, thus reducing its total volume to so small an amount that the systemic blood pressure falls. It is true that there is, in anaphylactic shock in this animal, an augmented escape of fluid from the blood especially in the liver and splanchnic area. There are several serious objections, however, to considering this to be sufficient in amount to cause the pronounced fall in blood pressure which characterizes canine anaphylactic shock. (1) Such an extreme loss of fluid from the blood should cause a marked concentration of the red blood cells in the peripheral circulation. This is either not found at all or at least not in sufficient degree to account for the violence of the reaction. (2) It is difficult to conceive how the capillary bed of one or more organs could, within the space of from two to five seconds, become so permeable as to allow such an amount of fluid to escape from the blood as to reduce arterial blood pressure from 120 to 40 or even to 30 mm. Hg. Even the most violent cases of pulmonary edema, in which the patient is in danger of drowning in his own fluid, develop more slowly than this is alleged to do. (3) Such a voluminous escape of fluid from the capillaries should manifest itself in a distinct, not to say marked, edema. This is either absent, or at least very slight, in a dog in anaphylactic shock.

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* Dale, J. Pharmacol. and Exp. Therap., iv (1912–1913), pp. 167 and 517.

† Shultz, Hygienic Lat. Bull., No. 80 (1912).

‡ Simonds, J. Am. Med. Assn., lxxiii (1919), p. 1437; Simonds and Arey, Anat. Record, xviii (1920), p. 219.

§ Manwaring, J. Immunol., viii (1923), pp. 191, 211, 217, 229 and 233.

‖ Peterson, J. Immunol., viii (1923), pp. 323, 349, 361, 367 and 377.

Anti-anaphylaxis.—An animal which has suffered anaphylactic shock from a second injection of foreign protein to which it is sensitized, will not respond to a third injection of similar protein. This refractory state is termed anti-anaphylaxis or desensitization. It persists for a considerable period of time, but subsequently disappears, however, and the animal again becomes sensitive. Anti-anaphylaxis can be produced (1) by re-injection of the homologous protein before the expiration of the incubation period, that is in less than 10 days after the injection of the sensitizing dose; or (2) by the injection of a small, sublethal dose given during the anaphylactic state. In the latter case the animal suffers a very mild shock and becomes refractory to a larger injection of the same kind of protein. No satisfactory explanation has yet been offered for the phenomena of anti-anaphylaxis.

Passive Anaphylaxis.—The serum of a sensitized animal when injected into a normal animal will render the latter sensitive to the homologous protein. The serum of a desensitized or anti-anaphylactic animal may also transfer sensitization. However, in both instances, some hours must elapse before the recipient becomes reactive. The offspring of a sensitized mother are also sensitized to the same protein as the mother. The passive transference of sensitization is one of the chief reasons for placing anaphylaxis in the group of immunologic reactions.

Anaphylaxis in Humans.—In about one out of 50,000 cases, the injection of antitoxic serum into humans results in sudden death. Most of these unfortunate events have occurred in persons who suffered from asthma. These sudden deaths are believed to be due to anaphylactic shock, although in the majority of cases the fatal reaction follows a first injection of the foreign serum without any apparent opportunity for artificial sensitization. Whether the reaction in the human is of the type seen in the guinea pig, the rabbit or the dog, or of some other type, has not been determined.

After the injection of a comparatively large amount of foreign serum, there frequently occurs, about the sixth to the fourteenth days, a reaction which has been called *Serum Disease* by von Pirquet and Schick.* This may be characterized by fever, rashes, urticaria, painful swellings of the lymph glands, edema, rheumatoid affections of the joints, and albuminuria. Serum disease is believed to be due to the failure of the body to completely excrete all of the foreign protein before the process of sensitization is complete. The retained protein serves as antigen and all of the constituents are therefore present to initiate the reaction.

A first injection of a foreign protein usually induces a condition of sensitization or allergy in humans as well as in animals. A second injection, if made between 10 and 40 days after the first, is followed by an *immediate reaction*. This resembles serum disease in all essential respects except in the promptness with which it follows the second injection. If the second injection is administered more than 40 days after the first, an *accelerated reaction* usually occurs. The symptoms appear after an abbreviated incubation period of from four to six days, and are of shorter duration than in the ordinary form of serum disease.†

In 1903, Arthus‡ reported that repeated subcutaneous injections at short intervals of foreign serum into rabbits resulted in edema, hyperemia, and later necrosis at the site of the injections. This has come to be known as "Arthus' Phenomenon." A somewhat similar reaction sometimes occurs in man during immunization against hydrophobia by Pasteur's method. These injections are made subcutaneously into the anterior abdominal wall. After the ninth to the twelfth day, there sometimes appears a painful edema and hyperemia of the skin and subcutaneous tissues of this region. This is not due to infection, but is a local reaction very like Arthus' phenomenon.

Diagnostic Skin Reactions.—The specificity of the tuberculin, luetin, mallein and typhoidin skin reactions for the diagnosis of tuberculosis, syphilis, glanders and typhoid fever, respectively, is based upon a condition of sensitization or allergy to the protein of the microörganisms which cause these diseases. Their mechanism is quite different from that of the Schick reaction by which resistance to diphtheria is determined. In the Schick test, a soluble bacterial toxin is injected into the skin. If antibodies for this toxin are present in the body fluids, they neutralize the poison and no reaction follows. In the tuberculin test, for example, bacterial proteins are injected into the skin. If specific antibodies are present, they split the homologous bacterial protein and liberate its poisonous portion, and a local reaction results. If the patient has not suffered an infection with the specific microörganisms, his blood and body fluids do not contain antibodies and no local reaction follows. A positive Schick reaction, therefore, indicates the absence of the corresponding antibody; a tuberculin skin reaction is due to the presence of specific antibodies. The skin tests for sensitization to pollens in hay-fever, and to different proteins in asthma have the same fundamental mechanism as that described for tuberculin.

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* Von Pirquet and Schick, "Die Serumkrankheit," Leipzig (1905).

† v. Pirquet, Arch. Int. Med., vii (1911), pp. 259 and 383.

‡ Arthus, Compt. rendu Soc. de Biol., lv (1903), pp. 20.

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